

A CLINICAL SURVEY
of a few cases of
PNEUMONIA
with special reference to
(1) Leucocytosis
(2) Coagulation time
(3) Leucolysis
(4) Idophilia
(5) Phagocytic activity

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INTRODUCTION

Chapter I Cases with charts of

- (a) leucocytosis
- (b) coagulation
- (c) Differential counts.

1. Normal
2. with empyema
3. with abscess formation
4. fatal
5. with Hemiplegia
6. with otitis and parotitis
7. with Taenia mediocanellata
8. fatal
9. Confluent Broncho pneumonia
10. Normal
11. with commencing necrosis
12. with phthisis
13. with Bronchitis
14. normal
15. normal
16. fatal
17. normal.

18. with syphilis
19. normal
20. mild case
21. with Jaundice.

Chapter II.

- (1) Leucocytosis in Pneumonia
 - (a) work of previous observers
 - (b) personal observations, with tables.
- (2) Differential counts in Pneumonia, with tables.
- (3) Coagulation times in Pneumonia, with tables.
- (4) Leucolysis in Pneumonia
 - (a) work of previous observers
 - (b) personal observations
- (5) Iodophilia applied to Pneumonia.

Chapter III.

The Phagocytic activity of the blood in pneumonia.

- (1) Introduction
- (2) Normal blood incubated with staphylococcus pyogenes aureus.

- (3) Normal blood incubated with Fraenkels pneumococcus
- (4) Pneumonic blood with Fraenkels pneumococcus
- (5) Pneumonic blood with staphylococcus pyogenes aureus.

Conclusion -

The fundamental principle of this Thesis is the careful comparison of a few of the changes noticed in the blood, with the general history, symptoms, etc. of a few cases of Pneumonia.

The blood in pneumonia is a subject that has been somewhat peculiarly treated, certain points, as the absence of a leucocytosis being the indication of a fatal termination, having a very large literature, of comparatively recent date. Others, as the rapidity of coagulation, a symptom of immense importance, and recognised since pneumonia was first known as a distinctive disease, have been seldom dealt with, and, as far as I have been able to find out, never critically compared with the other factors in the blood on the lines which I have attempted. Then the immense subject

of the action of these leucocytes (which are in such increased numbers, in the circulating blood), upon toxic organisms, when the blood is alive at blood temperature - has never been taken up by any observer.

In reading over the Literature of this subject, I was much struck by two things.

(1). The amount of contradiction shown in this work. In every branch of it, this is seen from the life history of a leucocyte, to its phagocytic power - And what can we expect, when observers like Gulland and Ehrlich differ fundamentally on the first, and Ehrlich and Metchnikoff on the second.

(2). The generalization of Pneumonia. That is, that every case of Pneumonia is looked upon as virtually the same as another without due regard to the complications and sequelae. An illness is said to be Pneumonia, the leucocyte counts are taken for a few days, and then the patient dies.- It is classified as lobar pneumonia but was the patient's death due to this alone, or to supervening gangrene, abscess formation with mixed infection, or any of the numerous complications

of this disease ? To take an example, we read that Iaksch had sixteen cases with leucocytosis of which three died, and five without, of which three died - That Ewing had a hundred and one cases, and of these six had no leucocytosis and all six died. Then we find Maragliano doubting this and saying that he finds cases dying with 40,000 leucocytes and often living with 4,000 - Now I think this difference is due very much to the tendency to slump cases. I have tried to counteract this by giving my cases separately and drawing attention, to any specialities that they may show. This has added greatly to the length of this paper, but I have been so struck with the want of accuracy, and tendency to dogmatism shown by many workers on the blood, that I hope the extra length of this Thesis will be excused.

I have also tried to get my cases as typical as possible having dealt only with those showing Fraenkels pneumococci in the spit, along with well marked physical signs. - I must exclude two exceptions - both being cases without spit at all, but having such a history and showing such well

marked physical signs as to leave no doubt in my mind, that they were acute lobar pneumonia.-

I would also draw attention to the fact that the conclusions drawn from this Thesis are not from the cases alone which I have followed from day to day, but also from a very large number of cases not specified.

I think we may take the normal number of Leucocytes to be 7000 - Tumas estimated them as 4800 to 9000, Iaksch as 7000, Reider as 7600, Halla and Boeckman as 4000 to 9000, Thoma and Lyon as 6700 to 10,000, Osler as 6000.

I am afraid that I have not space to take up the literature of the different varieties of cells, suffice it to say that - Wharton Jones based his classification on whether the leucocytes were granular or not.

Ehrlich started a classification with analine dyes.

Kanthack and Hardy modified this somewhat. Then Metchnikoff regarded them from their action on microbes.

The classification most used now is a modification of Ehrlich's, and it is this that I

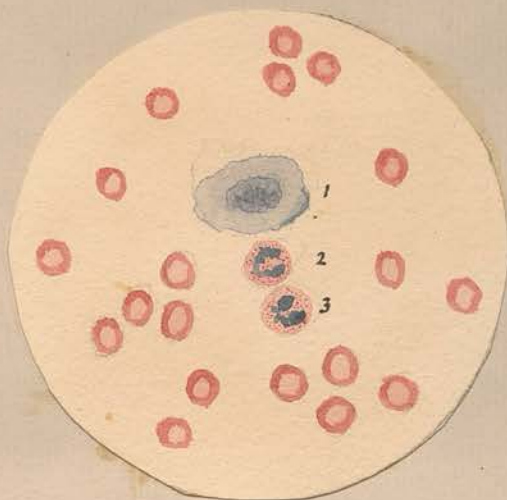
have adopted.

1. Small Lymphocytes.
2. Large Lymphocytes occasionally called hyaline cells.
3. Eosinophile cells.
4. Polymorphonuclear cells with faintly Oxyphile granules.
5. Transitional cells with a single nucleus staining somewhat darker than the large lymphocyte. It also is kidney shaped with a bay or indentation. It has scanty faintly oxyphile granules.
6. The myelocyte and its companion the oxyphile myelocyte.
7. The Basophile cell "mastzellen" whose granules are not colored by Ehrlich's triple stain, but brought out distinctly by Thyonin blue.

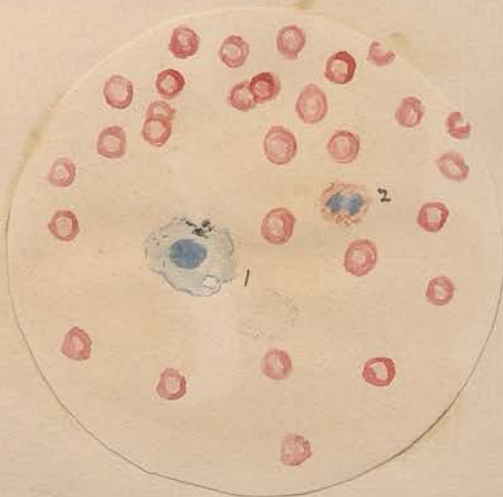
Now there is a cell which I have often seen after the crisis in penumonia but only in very small numbers.

Turk described cells he called Reizungsformen as occurring in pneumonia. These resemble the

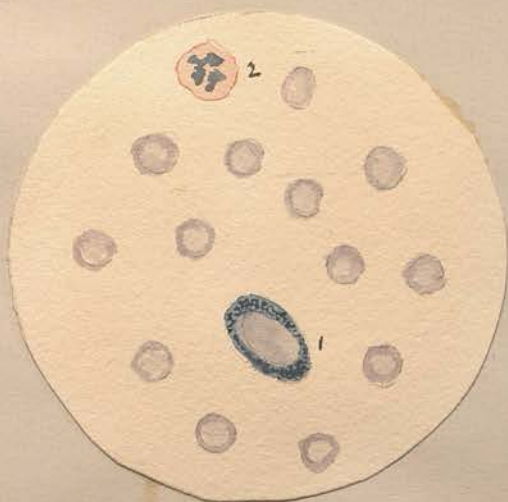
cells I have noticed, in several ways. I have never seen them in a fatal case and, never before the crisis. They are large cells. Often larger than the myelocyte. They resemble the large lymphocyte in that they have a single nucleus and basophile protoplasm as a rule. They seldom contain granules - in this differing from the myelocyte - The nucleus is centrally placed and as a rule stains darkly with basic stains. Their protoplasm is rather plentiful much more so, than in the myelocyte's and is often vacuolated. I have seen them in films with foreign matter enclosed in their protoplasm. In my incubation experiments to be enumerated later on, I found the large lymphocyte proper, not nearly so phagocytic as the polymorphonuclear. Now these cells mentioned above are much more so and I think Metchnikoff when he gave the name macrophages to large lymphocytes must have had these in his mind. On the injection of organisms into the peritoneum of a rabbit, it was proved by several observers that after a chemio-taxis of polymorphs, a number of large mononucleated forms appeared at a later time.



1. Large mononucleated cell
2. Polymorphonuclear cell
3. do.



1. Large mononucleated cell with enclosed foreign matter and vacuoles
2. Polymorphonuclear cell



1. Large mononucleated cell
2. Polymorphonuclear cell

They were so phagocytic, that not only did they seize upon the bacteria, but engulfed polymorphonuclear cells as well. These are the cells that I think correspond in the rabbit to the cells noticed by myself in pneumonia. They are evidently only found in the blood upon the neutralization of the toxin - and their chemiotaxis in this, differs from that, of the polymorphs which are attracted, either by the products of the bacterial life, or by the products of the destroyed leucocytes (Löwit) or as Horbacewski holds; the nuclein from this destruction. Türk believed the cells, he noticed, to be stimulation and immature forms, evidently an early stage of the myelocyte before granules had been developed. Engel also noticed this cell and calls it, by the useless name of mononuclear cell. Weil gives it the name of non-granular myelocyte.

My films, from which these facts have been obtained are stained in the following methods. -

1. Ehrlich's triple stain. It was some months ^{after} ~~from~~ my commencement of this subject before I was able to obtain a reliable brand of this stain.

2. Fixing with Formalin in alcohol and staining with watery solution of eosin and methylene blue.

3. Fixing with Hyrarg Perchlor and staining with an alcoholic solution of eosin and Loefflers methylene blue.

4. In a few instances I used Haematoxylin instead of methylene blue, in the second method.

The order I intend to carry through in this Thesis is -

Chapter I. Cases with post mortem reports and charts of temperature, leucocytosis, coagulation time and differential count tables.

Chapter II. A comparison of the above results.

Chapter III. The resistance of the blood to toxic organisms.

Chapter IV. Conclusion.

I must here mention my indebtedness to Dr. James, for his kindness in placing the cases of pneumonia in his ward at my disposal for observation.

Case I.

Age : 46

Sex : male

Occupation : glass blower

Place of Residence : Grassmarket

Admitted : Feb. 5th

Examined : do.

Complaint : General weakness and malaise with shortness of breath, cough and spit and pain in the right side.

Duration : Three days.

History : He shows no hereditary tendencies, as far as can be made out, but is uncertain regarding the cause of death of his parents.

Habits as to Food and Drink.

As he is very often in destitute circumstances, these are far from satisfactory. He seldom knows when he will get his next meal, and when he does happen to have any money spends it as often on alcohol as food. He indulges in this freely when ever he has the opportunity. He is also a considerable smoker.

Surroundings at Home.

His home is occasionally in a lodging house,

when he can afford it, at other times he sleeps out at night, in any cover he can find.

At work.

He is more often out of work than at it. There are a good many changes of temperature, the blowing of the glass subjecting him to many draughts.

Previous Illnesses.

Has always been fairly healthy, never suffering from any serious illness. He has never had pneumonia before nor influenza. Has never met with any accidents. No history of ~~Emphysema~~.

Present Illness.

He had been out of work for some time, living a very rough and ready life, and drinking as much as he could, up to Feb. 2nd. That night he slept out, all night, very cold, as he was poorly clad and the weather was cold, there being frost at the time. In the morning he was seized with a pain in his head, and severe shivering, his teeth chattering. It passed off a bit, but was succeeded by a pain in his side and a cough, and he came to the Infirmary seeking admission.

State on Admission.

He is a small man and looks thin and spare - His development and muscularity are poor. The body being poorly covered with fat. His face is thin and nervous looking with some what shrunken cheeks. There is no Jaundice or cyanosis. He is perspiring somewhat, and his face is flushed, especially in the malar region, neither side however being more marked than the other. He shows no herpes on his lips.

Temperature 104.

Respiratory System

The patient is lying on his right side with short shallow respirations 36 per min. and a slight grunting sound on expiration. The Breathing is regular, and costo abdominal in type. He says if he takes a deep breath it hurts him.

Cough.

This is very severe, he feels a bit of sputum which he wishes to bring up, and coughs hard to do so even though he complains of it being very painful.

Sputum.

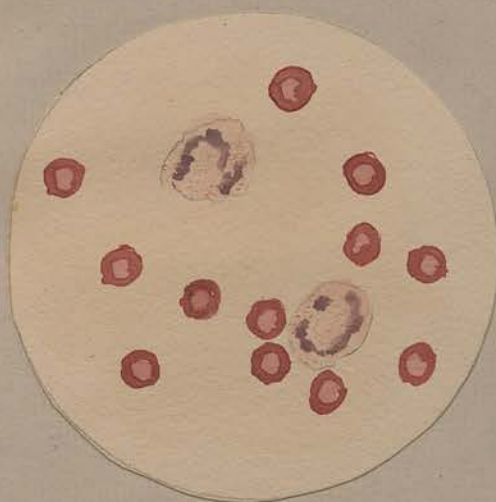
Sputum.

It is fairly copious and very tenacious. It is muco-purulent, and does not show the rusty color but is a pale yellow. It contains mucous cells, and many pus cells. The tremendous majority being polymorphonuclear and many, being in a more or less broken down condition. I looked carefully, but was unable to find any eosinophile cells. Upon staining with ~~Grahn~~ Grams method very numerous Fraenkels pneumococci were found, also associated diplococci, some of large size, and without capsules. These were not so numerous as the true Fraenkels also some cocci and mouth organisms. This was a somewhat peculiar case as many of the pus cells seemed to contain the diplococci, six diplococci being contained in one cell that was examined. I found this a very rare condition however, as very few of my slides show it. Voice low pitched and he speaks in a subdued voice.

Physical examination of Chest.Inspection.



SPUTUM FILM FROM CASE I



BLOOD FILM FROM CASE I

NUCLEI PERIPHERALLY PLACED
AND EXTENDED.



BLOOD FILM FROM CASE XVI

ONE DAY BEFORE
DEATH

NUCLEI CENTRALLY PLACED
AND CONTRACTED

The chest is well formed, and shows no hollow -
:ing under the clavicles. It is poorly clad
with muscle. The chest moves freely on
inspiration and neither anteriorly nor poster-
:iorly could any difference in movement be
ascertained.

Palpation.

No difference in expansion can be made out.
The vocal fremitus is normal over the chest,
except at the right base posteriorly, where
it is somewhat increased.

Percussion.

Normal except at rt base, where there is
marked dullness up to the lower border of
the seventh rib in the line of the lower
angle of the scapula. The dullness is
absolute.

Auscultation.

Breathing medium pitched bronchial breathing
over this area, and at its upper border
broncho vesicular, with crepitations at the
end of inspiration. A few ronchi are heard
all over the right lung and the breath sounds
are coarse and expiration slightly prolonged

in all areas posteriorly. In the 5th and 6th Interspaces at the anterior axillary line some fine friction is to be heard. The vocal Resonance is increased at the right base posteriorly.

Circulatory System.

Nothing abnormal was noticed. The heart was of normal size, and the sounds closed in all areas. Occasionally there was slight reduplication of the second sound.

Pulse.

Regular in time and force Rate 98 per min. It is rather low tension with a medium sized wave. This is somewhat rapid in its rise and fall. No dichrotism can be made out. The artery wall is slightly thickened and the two pulses are the same.

Digestive system.

There is no herpes on the lips. The tongue is furred and dry. He has no appetite, but is very thirsty. The liver is normal in size, and the border does not project below the costal margin in the mammary line.

Nervous System.

He is very sleepless, but is not at all delirious, otherwise normal.

Urinary System.

Color amber, Reaction acid, Sp gr 1028.

No trace of Albumen. Chlorides diminished.

Reproductive system.

Normal.

Haemopoetic System.

The spleen is not enlarged and there are no enlarged glands.

Red Cells

5th Feb. 4,500,000

8th Feb. 4,300,000

12th Feb. 4,200,000

Haemoglobin

5th Feb. 92%

8th Feb. 90%

12th Feb. 87%

Treatment.

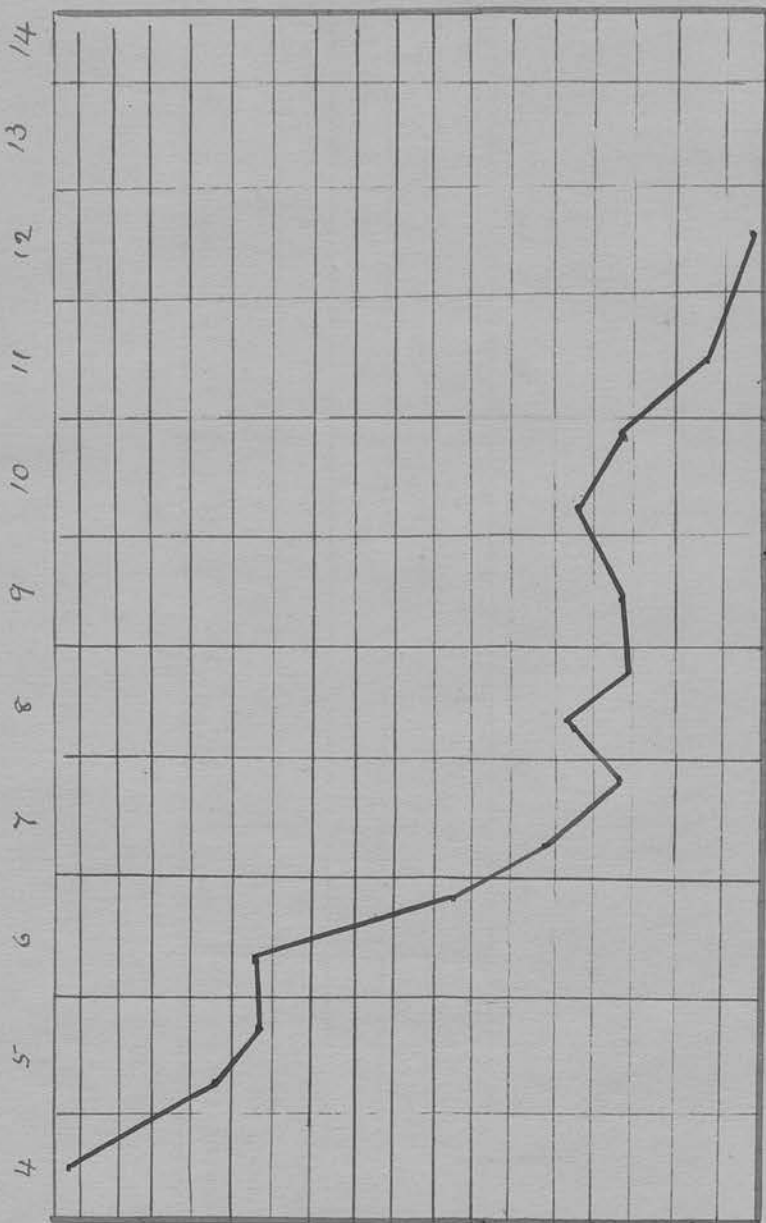
patient was kept on a milk diet and given

Pot Iod gr V

Spr Ammon Aromat $\frac{3}{4}$ ss.

Tr Camph Co a.a.m 20

14 day of illness



LEUCOCYTOSIS CHART.

42,000

40,000

38,000

36,000

34,000

32,000

30,000

28,000

26,000

24,000

22,000

20,000

18,000

16,000

14,000

12,000

10,000

8,000

8,000

↳ thousands of
leucocytes per cubic
millimeter.

15 day of month.

14 day of illness.

13

11

9

8

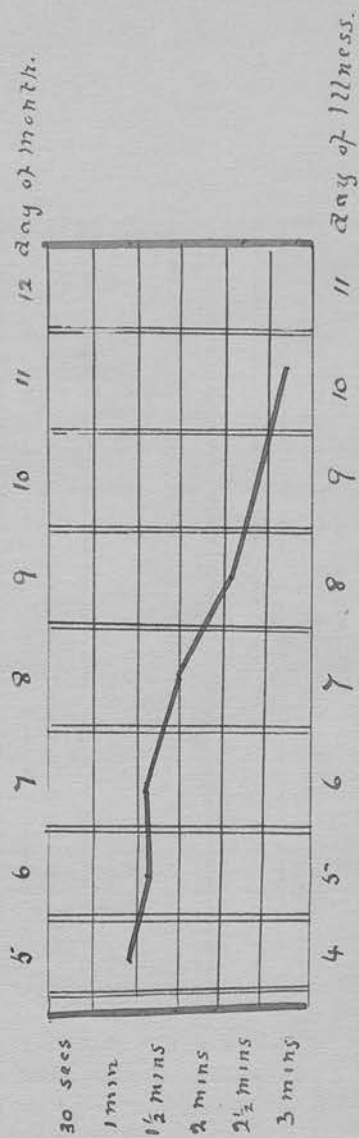
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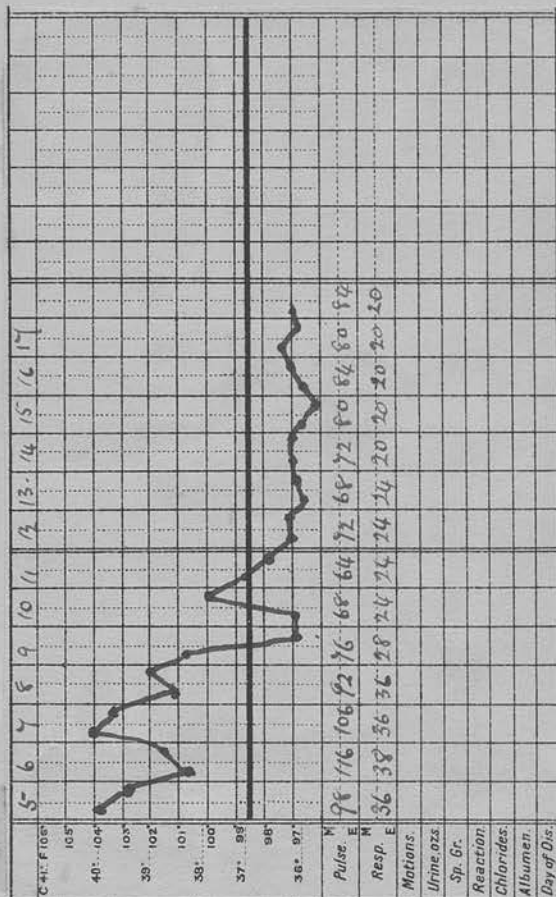
5

POLYMORPHONUCLEAR CELLS.	95	93	94	86	84.5	80	75-	-
TRANSITIONAL CELLS.	.7	1	1.5	1	-	.5	.8	-
LARGE LYMPHOCYTES	2.	3	2	10	9	8	9	-
SMALL LYMPHOCYTES	2.1	3	3	2.5	6	10	14	-
EOSINOPHILE CELLS	.2	-	-	.5	.5	1	1.2	-
MYELOCYTES.	-	-	-	-	-	-	-	-

DIFFERENTIAL COUNT TABLE.



COAGULATION CHART -



Temperature Chart.

Every four hours. On the seventh of Feb, as his pulse was rather weak, he was given Tinct Strophan m V every four hours. The ice bag was occasionally applied to relieve the severe pain in the right side.

Notes on the Case.

On the 8th the patient had his crisis, the temperature falling from 102 to normal. It was up to 100 the following day but on the 10th again fell to normal and remained there.

The physical signs on the 8th were more marked, the dullness having spread to 1 in above the angle of the scapula posteriorly. After this, resolution went on satisfactorily and the patient progressed rapidly towards recovery.

Case II.

Acute Lobar Pneumonia complicated by

Empyema.

Boy aged : 10

occupation : At school

admitted : Jan. 27th

examined : do.

Complaint : Cough, weakness, and pain in right side.

Duration : Two days.

History : Family.

Father and Mother alive and healthy. His brothers and sisters also are in good health and there have been no deaths in the family.

Habits as to food and drink.

He gets plenty of good food and takes it well.

Surroundings at Home.

He is quite comfortable. Lives in a flat in a pretty high situation and attends the Board School.

Previous Illness.

Except Measles and some childish ailments, he has had no illness always being a healthy boy. No accidents of any kind.

Present Illness.

On Jan. 25th he went down to the Public baths and being a good swimmer, he stayed a considerable time in the water. He blames his illness to this as he felt chilled and cold before leaving the baths. He went straight home, and was sent to bed where he felt hot and uncomfortable, and did not sleep well. The next morning he was seized with a pain

in his right side. He got worse and was sent in on Jan. 27th.

State on Admission.

He is a fairly well developed boy with moderate muscularity and a good deal of subcutaneous fat. He is not cyanosed, but flushed and shows no herpes. He is very restless, and inclined to cry if touched at all. He is lying on his back and occasionally throws his legs about.

Temperature 103.

Respiratory System.

He has short shallow respirations, 40 per min. Has no expiratory moan. He says he cannot take a deep breath without causing him great pain all over the right side. The pain is stabbing in nature. His cough is short and dry and is not frequent at all, and does not come in paroxysms.

Sputum.

He seems to have very little sputum and what he has, he always swallows instead of bringing it up.

He is very restless and difficult to examine.

Inspection.

The chest is well formed and clothed with muscle. The left side has more movement than the right side. This is especially limited at the right base posteriorly.

Palpation.

Confirms inspection. The vocal fremitus is more marked on the right side. He has a somewhat high pitched voice and it is not very distinct.

Percussion.

Anteriorly : There is a slight rise of pitch over the right lung anteriorly, left normal. Posterior : Marked dullness reaching to the spine of the scapula.

Auscultation.

Over this dull area, loud bronchial breathing is heard: Some fine crepitations are made out near the upper border. Above this the breathing is broncho vesicular. On the left side conducted bronchial breathing is heard.

Bronchophony is heard almost all over dull area.

Slight medium friction is heard at the anterior margin of the dullness on the mid axillary line.

Circulatory System.

The heart is not enlarged, and the sounds closed in all areas. The pulse is 132 per min. regular in time and force. The tension is low and the wave, of small volume, and very rapid in its rise and fall. No dichrotism can be made out.

Integumentary System.

The skin is dry, no rash is to be seen anywhere and no herpes upon the lips.

Alimentary System.

The tongue is dry and coated with a white fur. No sordes on the teeth. He has no appetite and his bowels are apt to be confined, otherwise normal.

Nervous System.

Patient is restless and a little apt to be delirious at night.

Urinary System.

Color amber sp gr 1032. Reaction acid.

A trace of albumen. The Chlorides diminished.

Reproductive System:

Normal.

Haemopoetic System:

There are no enlarged glands. The spleen is slightly enlarged.

Treatment.

Patient was given Spr ammon aromat and Spr aether nit along with Tr Camph Co m XV. every four hours. He was sponged down from time to time when his temperature rose above 103. The ice bag was applied to the chest to relieve the severe pain in side. On the 28th Jan. he was given Tr Strophan mV. every four hours.

Progress.

28th Jan. He was sponged and this brought his temperature down from 104 to 101. His pulse was 122. That night he was rather delirious.

29th Jan. Some herpes appeared on the

upper lip.

2nd Feb. He seemed rather better, the temperature still remaining at 103. The dullness at the right base is very marked, and the breath sounds not so distinct. V F and V R are somewhat diminished from what they were.

His cough was very troublesome coming more in paroxysms. No spit has appeared yet.

4th Feb. The breath sounds were very faint at the right base, and fluid was diagnosed. A large sized hypodermic was inserted and some fluid drawn off. It was sero purulent of a light yellow color and not blood stained. From it some pure cultures of Fraenkels pneumococci were obtained. Microscopically the fluid contained numerous polymorphonuclear cells. Some broken down and very numerous Fraenkels pneumococci. They were not enclosed in the cells. Eosinophile cells were not present, as far as could be made out. His temperature is gradually falling and he seems a good deal easier.

14th Feb. His temperature has been rising and developing a swing of 2 to 3 degrees, so it was decided to call in surgical assistance. Mr Caird made an opening, and inserted a drainage tube. A quantity of pus was removed IV - VI. Fraenkels *pneumococci* were still present but with *staphylococci* associated with them. After this opening his temperature came down and he progressed steadily towards recovery.

The Blood.

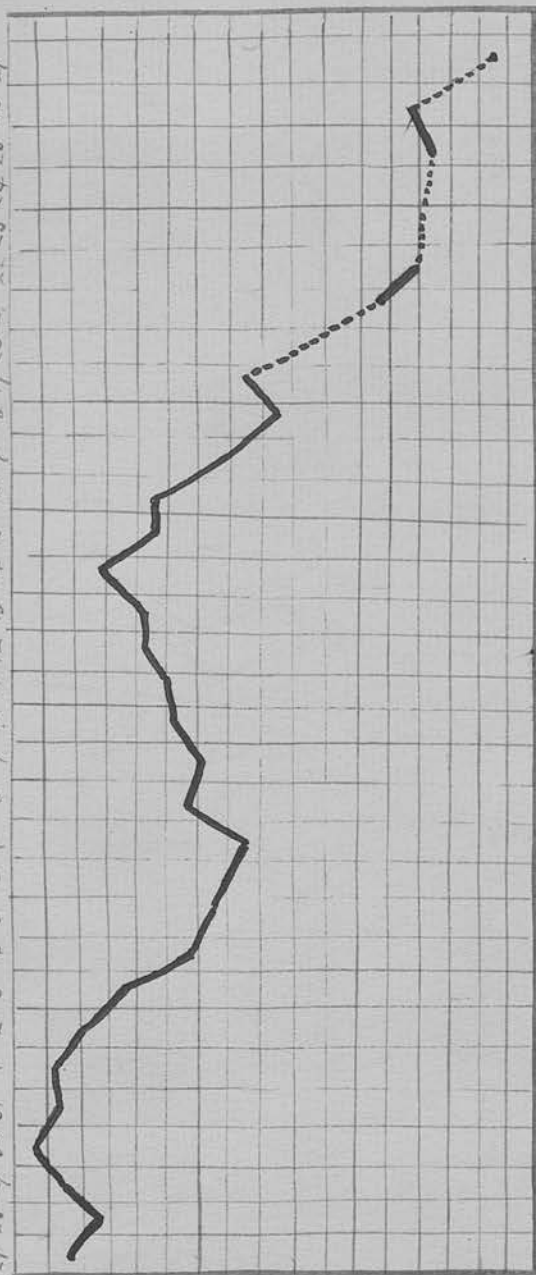
On the 27th Jan. the red cells were normal in number, well formed and evidently of normal size.

The Haemoglobin was 87 p.c.

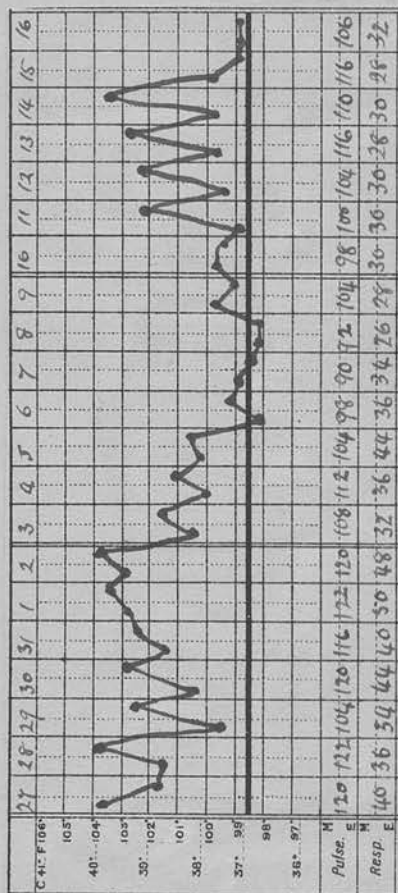
On the 3rd Feb. at the time of the crisis the red cells were somewhat reduced, being 4,200,000 and the Haemoglobin 74 p.c.

The count of the red cells was not again taken until the 15th Feb. when they had risen to 4,340,000 and the Haemoglobin 80 p.c.

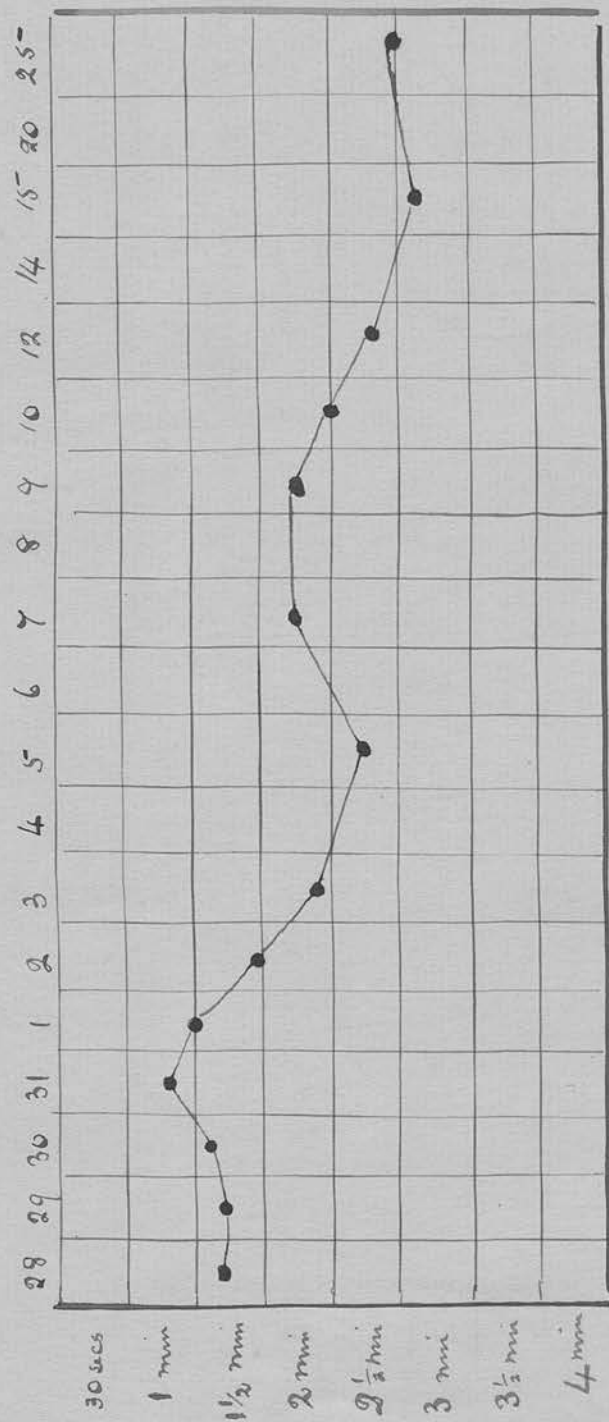
2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 day of illness.



LEUCOCYTOSIS CHART,



TEMPERATURE CHART,



	28.	30	1	2	3	4	5	7	9	10	12	15	16	20
POLYMORPHONUCLEARS	76	75	70	78.5	75	79	80.5	78	81	80	77	80.2	79	70
TRANSITIONAL	4	1	6	4	2	3	2.1	3.3	1.5	2.1	1.5	2.5	.5	1
LARGE LYMPHOCYTES.	14	20	17	12	10.1	13	12.4	13	12	10	13	10	7	9.4
SMALL DO.	5	3	6	4.3	12.2	4	4	5	3	5	7	4.3	12	16.5
MYELOCYTES.	1	1	.7	1	.7	.6	.5	.4	2	1.4	.2	.5	.5	.5
EOSINOPHILE CELLS.	-	-	.3	.2	-	.3	.5	.2	.6	1.5	1.3	2.6	1	2

1. 2.

1. First incisions,
2. Second incisions, as tube too small in size.

DIFFERENTIAL COUNT TABLE.

Case III.

Man aged : 33

occupation : labourer in gas works.

residence : 4 Hill Place

Date of Admission : 3rd Feb.

Examination : do.

complaint : Severe cold

Duration : four days.

History : Hereditary : good

Food and Drink:

He is well fed and does not drink to excess.

General surroundings:

Married man with two healthy children.

They live in a one roomed house.

At work :

This consists of filling tanks with Ammonia water. A most irritating occupation and much exposed to draughts.

Hours 4 a.m. to 5 p.m.

Previous Illnesses :

Measles as a child, otherwise healthy.

Present Illness.

Commenced on Friday, with a Rigor which

lasted till he got home. He had great headache and pain in the small of his back. Also between the shoulders. He took some rum, but vomited immediately. He got worse and noticed his spit rather reddish, and was sent in on Feb. 3rd.

State on Admission.

Height : 5 ft. 7½ in.

Weight : 10 stone

The development and muscularity are good and there are no obvious morbid appearances. No cyanosis nor Herpes.

Temperature 101.

Respiratory System.

Breathing 28 per min.

Is regular and costo abdominal in type. He has no pain on breathing.

Cough : Short and irritating

Sputum : Not great in amount. Rusty in color and very viscid. Contains Fraenkels pneumococci and numerous polymorphonuclear cells in various conditions of breaking down.

Patient is a little hoarse when talking and talks in a low voice.

Inspection.

Expands better on left side.

Palpation:

Vocal fremitus is increased on the right side posteriorly.

Percussion:

Anteriorly marked tympanitis, more so, on the right side. Posteriorly marked dullness reaching from the base of the lung to the angle of the scapula.

Auscultation:

Marked bronchial breathing over this dull area with a few fine crepitations at the end of inspiration. Vocal resonance does not seem much increased. No friction is to be heard.

Circulatory system:

Heart slightly enlarged. The apex beat being in the 6 and 7 interspace. The first sound accentuated. A slight systolic and diastolic murmur can be heard at the aortic.

Pulse:

rate 120 per min. regular in time and force, with a large wave, rapid rise and fall. Poor tension. The artery wall somewhat thickened

and tortuous.

Alimentary System:

Lips cracked and dark patches.

Teeth sordes. Tongue furred. Appetite poor and great thirst. Otherwise normal.

Liver slightly enlarged, extends one inch below costal margin in the mammary line, and is tender on percussion.

Nervous system:

Restlessness, sleeplessness and slight delirium at night.

Urinary System:

Wine color bright amber, reaction acid, specific gravity 1018. Deposit of mucous.

Haemopoetic System:

No enlarged glands. Spleen and Thyroid are not enlarged.

Red cells 4,800,000

Haemoglobin 81 p.c.

Other systems normal.

Treatment.

Spr. Ammon Aromat. Spr Aether Nit and Tinct Strophan mV every four hours. Whisky every four hours.

Progress:

The temperature gradually fell and touched normal on the 8th February. After this it never rose above 99° till his death on the 11th. His pulse 127 per min. on the 3rd kept up its rapidity all through. It however became weaker on the 9th. The strophantus and whiskey were doubled. The pulse however got weaker. The physical signs remained pretty constant but some dullness was found on the 7th at the left base.

Post Mortem Report:

A fairly well developed man with some lividity posteriorly, greenish discoloration over thorax and abdomen.

On opening only very little pericardium can be seen due to emphysema.

Left Pleura : adhesion over posterior surface of lower lobe.

Right Pleura : adherent latterly and posteriorly.

Heart weight : 9 oz.

Aortic Valve not competent. The segments, thickened in the centre of the cusps.

The left ventricle shows distinct fatty change. The right ventricle and left auricle contained large pale thrombi.

Left Lung:

Weight 15 oz. general appearance markedly emphysematous. The lower lobe posteriorly being deeply congested and showing haemorrhages in its substance.

Bronchi congested and contain mucus and blood.

Right Lung :

Weight 3 lbs. 11 oz. The lower lobe was very voluminous, the middle lobe adherent to it, slightly infiltrated along its detached part and the rest emphysematous.

Bronchi contain thick mucus - pus
Lung Tissue.

Upper lobe : Emphysematous

Middle lobe : partly consolidated in stage of red hepatization with brownish spots where the haemorrhage had taken place.

Lower lobe : The pleura was stripped off and a large cavity was found filled

with thick muco pus deeply stained with blood, and of red jelly like appearance. On section shows dark patches and congested towards the outer part. When scraped thick muco pus was obtained. The lung tissue was said to be in a state of breaking down and suppurating.

Liver:

full of blood and shows some fatty infiltration and distinct cloudy swelling.

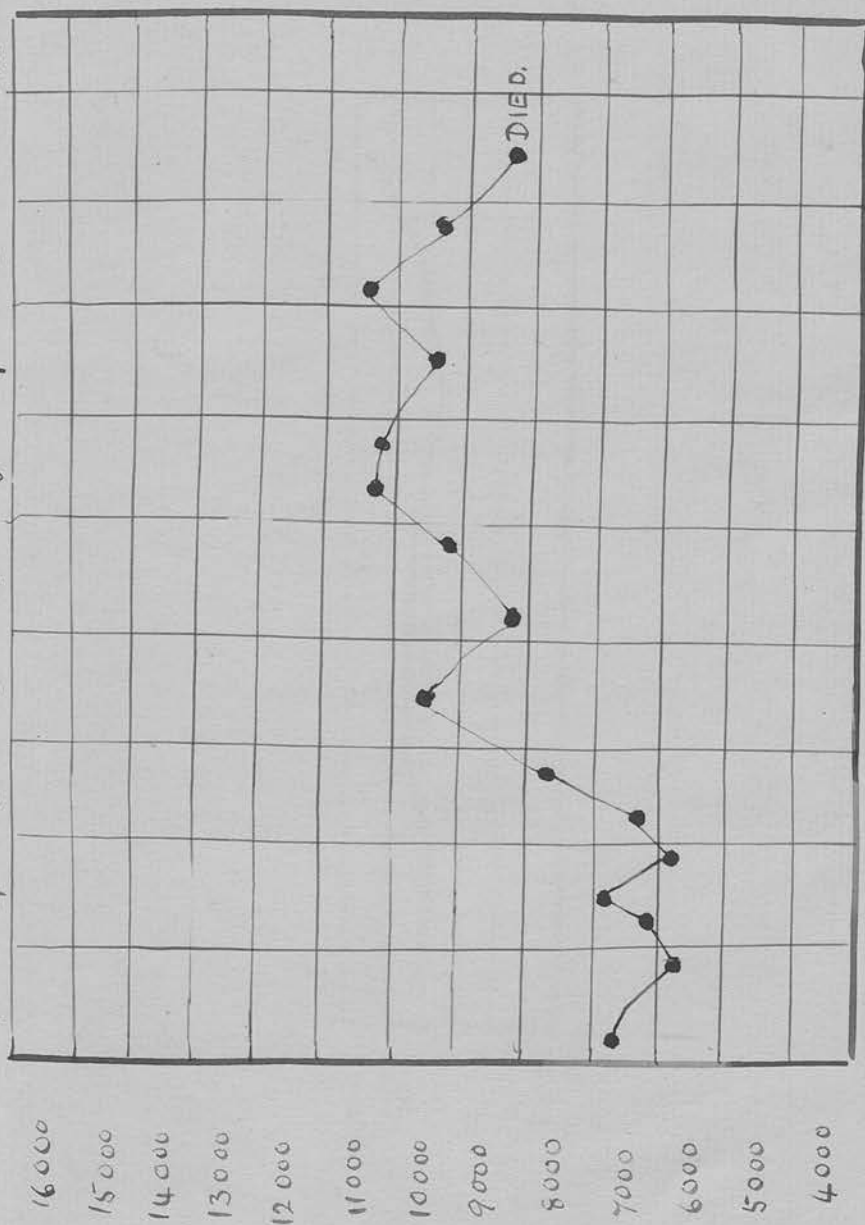
Spleen :

Weight 7 oz. pale and shows small haemorrhagic points.

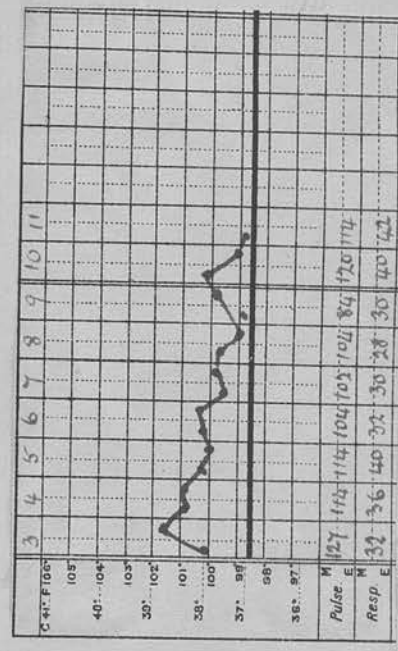
Kidneys:

Somewhat congested.

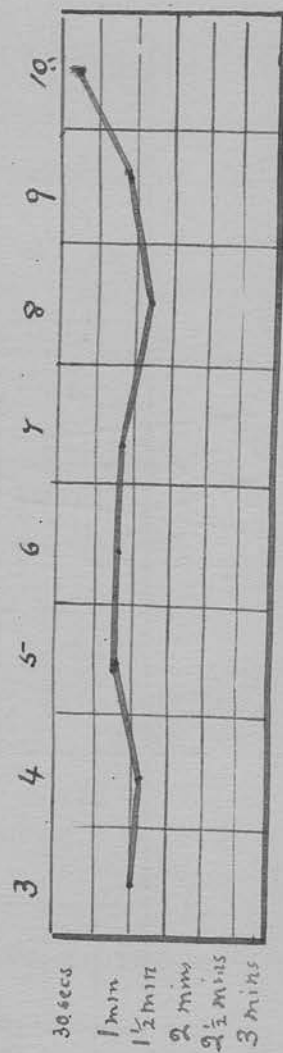
3 4 5 6 7 8 9 10 11 12 day of illness



LEUCOCYTOSIS CHART.



TEMPERATURE CHART



	3*	4	6	7	9	10.
POLYMORPHONUCLEARS	90	88.3	90	89	88	91
TRANSITIONALS	1	2	1	1	1	.4
LARGE LYMPHOCYTES	6	7	6.8	6.5°	10°	6°
SMALL LYMPHOCYTES	3	2.5-	1.7	3.5-	2	2
MYELOCYTES	-	.2	.3	-	-	.3
EOSINOPHILES	-	-	.2	-	-	-

* LEUCOCYTES ON THREE FILMS WERE COUNTED, 700.

o LARGE LYMPHOCYTES SHOWED VERY DARK STAINING OF NUCLEUS AND PROTOPLASM.

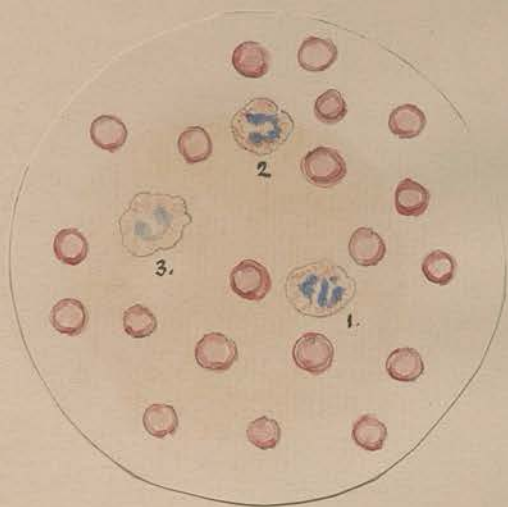
DIFFERENTIAL COUNT TABLE

	3	6	10.
BILOBED.	10	14	16
HORSESHOE	* 52	46	55
MULTIPARTATE	38	40	29

* NUCLEI VERY PERIPHERALLY PLACED.

SHAPE OF NUCLEUS.

SEE DIAGRAM OVER.



SHAPE OF NUCLEUS

1. MULTIPARTATE
2. HORSE SHOE
3. BILOBED.



1. LARGE LYMPHOCYTE
2. MYELOCYTE.

Case IV.

Man aged : 41

occupation : discharged soldier, who for the
last six months has been working
as a labourer.

admitted : January 9th

examined : do,

Complaint : severe pain in right side of chest
with short cough and spit.

Duration : nine days.

Family History: good

Habits as to Food and Drink:

He served as an Artilleryman in South Africa,
and says that on the whole, he was well fed
but had few chances of drink. Six months ago
he came home. He had no money saved and took
to work as a labourer. He drank heavily and
was often out of work. His food was poor and
he lived in a lodging house. He has been
living a rough life and has often been out all
night, in this country - but there is no
history of this shortly before the attack.

Previous Illness:

Always been a healthy man and does not acknow -

:ledge any venereal disease.

Present Illness:

This attack began nine days ago. He had been drinking hard and felt chilled. He had a severe fit of vomiting, and went to bed. For two days he remained in bed feeling cold and generally wretched. On the third day he felt a slight pain in his side. He gradually got worse and was admitted on the 9th of January.

State on Admission:

He is a strong well developed man.

Height : 5 ft. 10 in.

Weight : 12 stone.

He has a malar flush and some herpes on his lips.

Temperature 100.6

Respiratory System:

Respirations are 28 per min. regular and shallow and he complains of a very severe pain in the right side which catches him if he draws a long breath. This is worse anteriorly and spreads round to the back.

Cough : This does not trouble him much but is short and very painful.

Spit : Very tenacious and of a greenish yellow color. It is muco-purulent and contains many Fraenkels pneumococci.

On Physical Examination.

Nothing could be made out except slight friction on the right side anteriorly more marked in the fourth Interspace. No dullness was to be made out.

Circulatory System.

The heart is to all appearance normal. The pulse is 104 regular in time and force, with moderate tension and a fair sized wave. The vessel wall is a little thickened.

Haemopoetic System.

No enlarged glands." Spleen slightly enlarged and thyroid normal.

Alimentary system.

Loss of appetite, great thirst, furred tongue, no sordes, and otherwise normal.

Urinary System:

The urine is somewhat reddish in color, is alkaline in reaction with a specific gravity

of 1024. It contains a little blood and
3 .06 grains per oz. of albumen.

Nervous System.

He is very restless and inclined to be
slightly delirious at night, otherwise normal.

Treatment.

He was given Pot. Iodid gr V and Spr Aether
Nit and Spr Ammon Aromat M 20 every four
hours. He was also given Paraldehyde at
night. He was kept in bed on a milk diet etc.

Notes on the case.

On January 11th, Two days after admission
slight dullness was noticed just over the root of
the right lung, accompanied by bronchial breathing.
His temperature was 104 and his pulse 100.

On January 12th. He was worse, his tempera-
:ture being 103 and pulse 104 but somewhat weakened.
The dullness was much more marked. It had spread
over the whole right lung with bronchial breathing
increased vocal resonance and fine crepitations.

On January 13th. He was delirious all
night and somewhat wild, he was removed to No.6
ward. He was put upon Tinct Strophanthus M V.
every four hours and given a tablespoonful of

whisky every three hours.

On the 15th he was much weaker. His pulse 140. very feeble, and with a very small wave and a running character. He was quite unconscious and somewhat excited. Through the night of the 14th his pulse was often so rapid that it could not be counted, but he rallied somewhat in the morning and after this he gradually got weaker till he died at 5 p.m.

Post Mortem Report.

He is a well developed man with good nutrition. Greenish discoloration is present in the lower abdomen.

Left Pleura : old adhesion on diaphragmatic surface.

Right Pleura : old adhesions to the lower lobe.

Heart : A small milk-spot is present.

Right auricle contains a mixed thrombus passing into the right ventricle. There is a large pale thrombus extending into the Pulmonary artery from the Right ventricle.

Left auricle contains a mixed thrombus

extending into the Pulmonary veins.

The aorta contains a large antemortem clot.

The cusps are normal and the vessel fairly atheromatous.

Left Lung : Patechial haemorrhages under the Pleura at posterior part of lower lobe, above this, the lung is emphysematous.

Bronchi are congested and contain mucus.

The Pulmonary vessels contain thrombi.

The upper lobe is emphysematous.

The lower lobe shows increased friability, deep congestion and oedema.

Right Lung : Weight 4 lbs. 8 oz. yellowish lymph is seen between the Patechial haemorrhages particularly at the upper lobe beneath the Pleura.

Bronchi, deeply congested and show bronchitis and the Pulmonary vessels contain thrombi.

Upper lobe, congestion and oedema with consolidation inferiorly.

Middle lobe, grey hepatization with no pus.

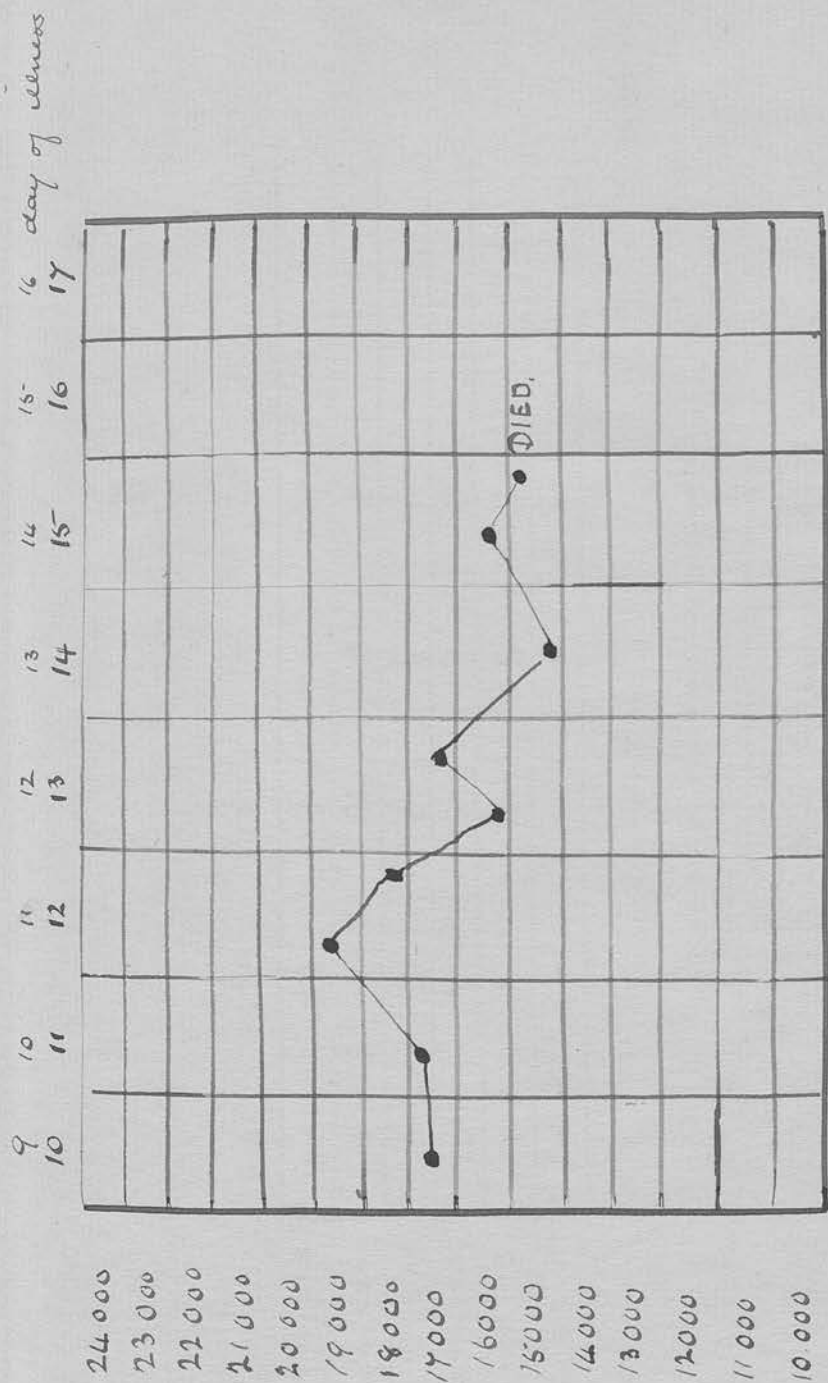
Lower lobe, shows grey hepatization.

The liver shows cloudy swelling and recent thrombi

in all the important veins.

The Spleen weighs 7 oz. and is somewhat diffident.

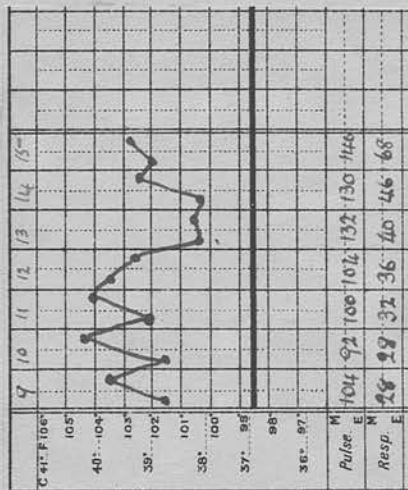
The Kidneys. These showed some cloudy swelling and congestion, and the Right Kidney some fatty change.



LEUCOCYTOSIS CHART

	10	11	13	14	15-
POLYMORPHONUCLEAR CELLS	80	80	78.5	76.7	79.
TRANSITIONAL CELLS	2	1.5	2.1	3	2.5
LARGE LYMPHOCYTES	6.2	8	9	8.3	10.3.
SMALL LYMPHOCYTES	10	9.5	8.4	9	6.2
MYELOCYTES	1.8	1.	2	3	2
EOSINOPHILES	-	-	-	-	-

DIFFERENTIAL COUNT TABLE



TEMPERATURE CHART

CASE V.

Man, Aged : 64

Occupation : Shoemaker

Place of Residence : Edinburgh

Date of Admission : February 13th

Examination : do.

Complaint : Pain in left side, shortness of
breath and paralysis of left arm
and leg.

Patient's Family History and Surroundings are good.

He has a good workroom and is not much exposed.

He has always been a temperate man.

Present Illness:

Five weeks ago just before the New Year, he had an attack of dropsy. His eyes being swollen in the morning, some swelling of both ankles and pain in the lumbar region. He was kept in bed and on milk diet. He recovered and was at his work for a day or two and then had a shock of apoplexy. He says that his left arm was quite useless and his speech slightly affected. On Monday the 10th he

had another stroke that is three days ago. In this the left leg was also included, his speech however was no worse. He was confined to bed and was seized with a severe pain in the left side and great shortness of breath on the least exertion and was sent in to the Infirmary.

State on Admission:

Patient is in a somewhat collapsed state lying on his back. He is a fairly well developed man. He shows no morbid appearances but slight cyanosis of the lips. His complexion is sallow, and there is no malar flush, nor any herpes. The paralysed left arm is kept folded on chest.

He is too ill to be moved much on examination.

Temperature 102°

Respiratory System.

His breathing is short and shallow but regular 40 per min and costo abdominal in type.

Cough is short and occasional and causes him great pain in the left side.

Spit is very tenacious and of a yellowish

color not rusty. It is muco-purulent. Shows the usual large amount of polymorpho - :nuclear cells and absence of eosinophile cells and is crowded with Fraenkels pneu - :mococci. It also, as so many pneumonic spits do, shows cells, resembling the diplo - :cocci of pneumonia, but often larger and without capsules. His voice is sometimes slightly indistinct, but he speaks quite sensibly and chooses his words well.

Inspection.

The chest is well formed and is well covered with muscle. The right side moves somewhat better on respiration than the left.

Palpation.

Confirms this and shows that the vocal fremitus is very much increased at the left base and somewhat increased at the right base posteriorly.

Percussion.

Anteriorly the Right lung is normal but there is slight tympanitis over the left lung.

Posteriorly.

Right Lung: there is some dullness posteriorly extending from the lower border upwards for three inches, otherwise it is normal.

Left Lung: complete dullness over the whole of the lower lobe and slight tympanitis over the upper lobe.

Auscultation.

Right Lung: medium pitched bronchial breathing is heard all over the right lower lobe and a few fine crepitations. Vocal Resonance is increased.

Left Lung: loud medium pitched bronchial breathing over the left lower lobe and medium crepitations at the end of inspiration. The Vocal Resonance is very much increased. At the upper border of the lower lobe some medium friction is heard extending anteriorly to the mid axillary line.

Circulatory System.

The heart is normal in size and the sounds are closed. There is slight accentuation of the second sound in the aortic region.

Pulse.

Is regular in time and force. The tension is good. The wave small and the vessel feels full of blood. The rapidity is 140. The vessel wall is a good deal thickened and is rather tortuous.

Nervous System.

This shows the usual symptoms of hemiplegia and is otherwise normal.

The Integumentary System.

No herpes is present, no oedema is now to be noticed.

Alimentary System.

The usual symptoms in this disease of want of appetite, furred tongue, etc. otherwise normal. The liver is not enlarged and the lower margin is covered by the ribs in the mammary line.

Urinary System.

The urine is amber in color. Reaction acid. Specific gravity 1030 contains a deposit of mucus and a trace of albumen. The Chlorides are diminished.

Haemopoetic System.

No enlarged glands and the spleen and thyroid are normal in size.

Treatment.

Spr Ammon Aromat and Spr Aether Nit M X every four hours. Also Tinct Strophanthus M V every four hours. Whisky was given every two hours.

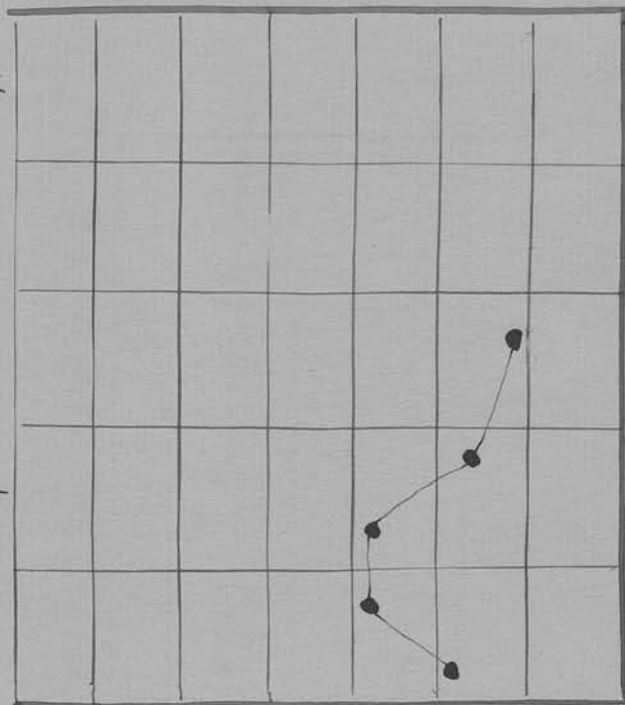
Progress.

On the 14th he was worse and complained of the pain in his side being very bad. The ice bag was employed and brought considerable relief. His pulse was no weaker and was 116. On the 15th as far as could be made out in his weak condition, the physical signs had not changed much. The dullness on the right side was however somewhat larger in area. At seven o'clock at night when lying on his back, he suddenly died.

No Post Mortem Examination could be obtained.

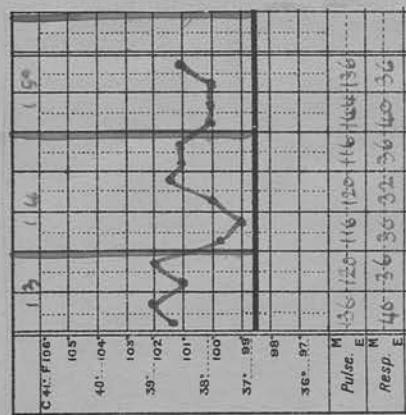
3 4 5 6 7 day of illness.
13 14 15 16 17.

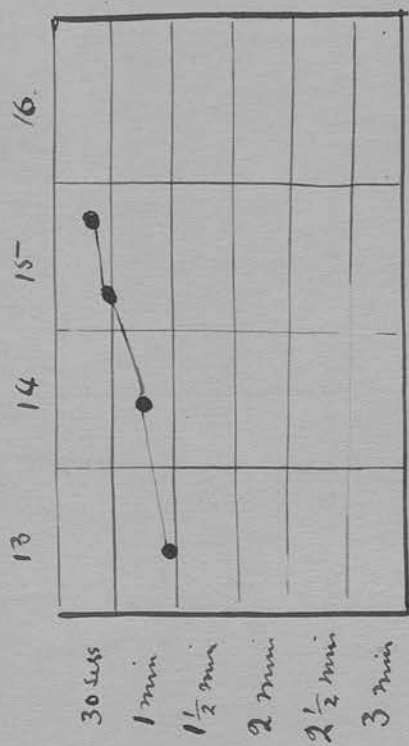
14,000
13,000
12,000
11,000
10,000
9,000
8,000



LEUCOCYTOSIS CHART

TEMPERATURE CHART.





COAGULATION CHART.

	13	14	15-
POLYMORPHO NUCLEAR	92	-	90
TRANSITIONAL	2	-	1
LARGE LYMPHOCYTES	2	-	6
SMALL LYMPHOCYTES	3.3	-	3
MYELOCYTES	.7	-	-
EOSINOPHILES	-	-	-

DIFFERENTIAL COUNT TABLE

BILOBED.	10
HORSESHOE	48
MULTILOBED	42.

SHAPE OF NUCLEUS.

CASE VI.

Man, Aged : 22

Occupation : Coalman

Residence : 104 Fountainbridge

Admitted : February 2nd.

Examined : do.

Complaint : Pain in right side with a
cough and spit.

Duration : Three days.

Family History : Good

Personal.

He leads a pretty exposed life walking round the streets in all weathers shouting "coals". His home is comfortable and he gets plenty of food. He indulges pretty freely in alcohol.

Previous Illnesses.

Those of childhood otherwise he has been healthy. No history of suppurating ears could be obtained.

Present Illness.

This began on January 30th, when he was at

a friend's house. He felt shivery, upset and so ill that he did not feel fit to go home. He stayed there all night and went home the following day. He got worse and was admitted on February 2nd.

State on Admission.

He is a strong well developed man slightly flushed but no herpes. There were no obvious morbid appearances.

Temperature 102.

Respiratory System.

His respirations are regular, rather shallow costo abdominal and 40 in rate. He complains of a breath being painful over the right side of the chest anteriorly.

Cough: This is severe and he says strains him very much.

Spit: This was rusty very tenacious and mucopurulent. It contained Fraenkels pneumococci.

Physical Examination.

This showed he had a well formed chest with the left lung moving more freely on respiration

than the right.

Palpation

Confirmed this. The vocal fremitus was slightly increased on the right side but not markedly so.

Percussion.

Complete dullness over the whole of the lower and middle lobes of the right lung. The upper lobe was slightly tympanitic.

Auscultation.

Loud medium pitch bronchial breathing is heard all over the dull area, except at the base where for a space of three inches from the lower border of the lung the breath sounds are much weaker. Medium crepitations are heard at the end of inspiration. The Vocal Resonance is much increased over the dull area but like the breath sounds is diminished in the lower three inches. Over the painful area in the 5th Interspace anteriorly there is marked medium friction.

Circulatory system.

The heart is normal in size and the sounds

closed. There is slight reduplication of the second Sound.

Pulse.

Regular in time and force rate 116. The tension is a little low. Artery wall is not thickened.

Alimentary System.

The tongue is furred and dry at the side. His appetite is poor and he is very thirsty. Bowels confined.

Nervous System.

Slight delirium at night and the ward is occasionally roused by a shout of "coals".

Urinary System.

No sample could be obtained as he passed his water involuntarily.

Integumentary System.

He perspires freely there is no herpes to be seen.

Treatment.

He received Pot Iodid gr V Spr Ammon Aromat M 30 and Tr Camph Co M 30. Milk diet, kept in bed and ice bag applied.

Progress.

4th February. His temperature is 103 and Pulse 120. Slight cyanosis and a little herpes on lips. He was given oxygen to inhale.

7th February. He had his crisis and his temperature dropped to normal and pulse to 84. On this day a little pus was noticed coming from the right ear. It was syringed with boracic lotion.

8th February. Considerable swelling of the left parotid gland was noticed. His temperature rose to 100. He had marked "Bells Paralysis" of the left side of face.

9th February. Pus was noticed coming from left ear. The discharge from the right ear had ceased. The swelling in the parotid gland was larger.

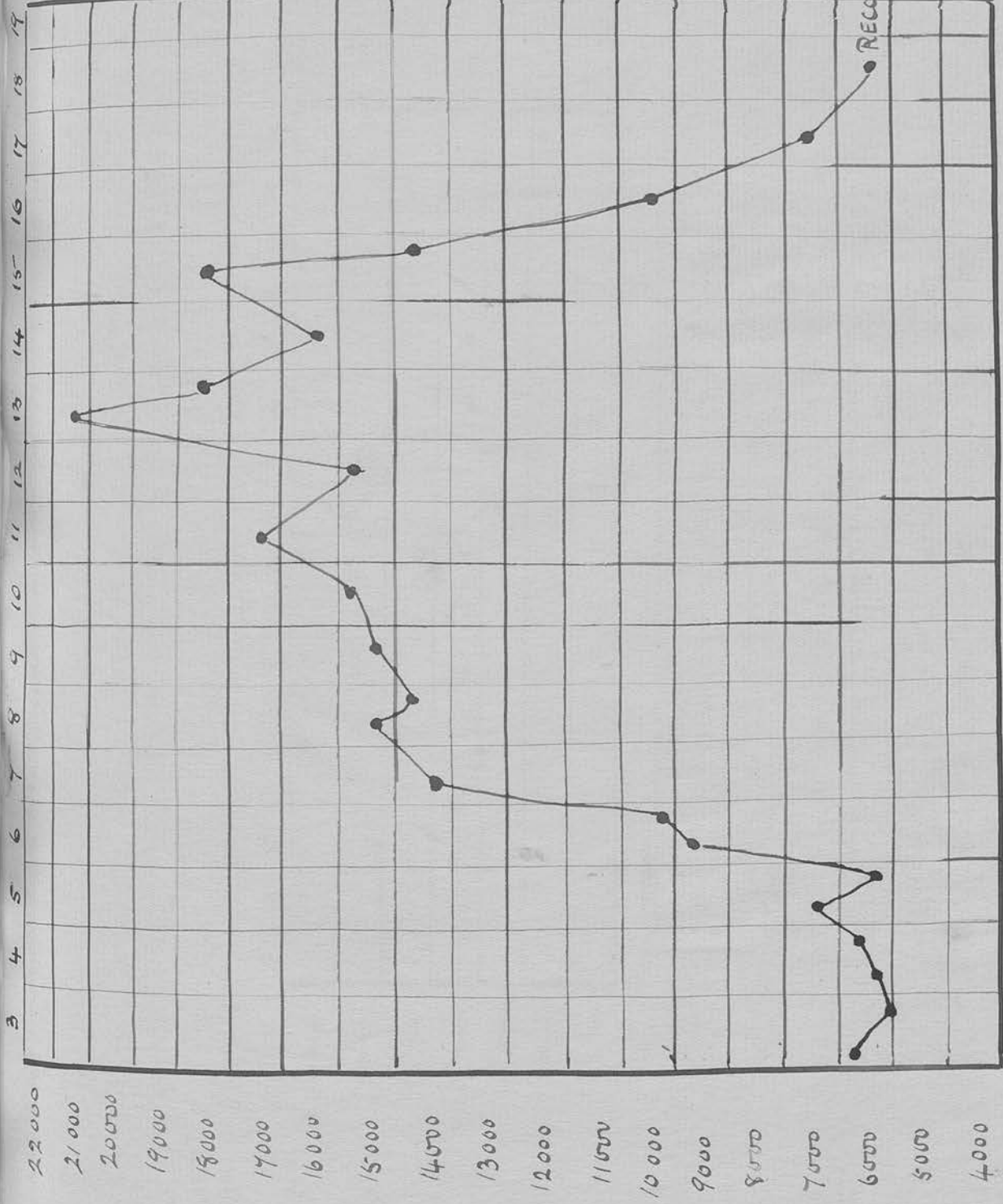
11th February. An incision was made into the enlarged gland but no pus was found.

13th February. An incision was again made by Mr Caird and one ounce of yellowish green pus containing staphylococci pyogenes aureus evacuated.

15th February. I enlarged the opening at

night and inserted a drainage tube. A large quantity of pus was coming from the left ear and upon syringing there was free communication between the opening in the gland and the ear. After this his temperature fell and he progressed satisfactorily. The Bell's Paralysis with immobility of the face on the left side, with want of closure of the eye, and drooping of the lower lid etc. remained. I could not find any loss of taste in the anterior part of the tongue. On the 15th of March when I saw the patient he was quite recovered from the Pneumonia, parotitis and otitis and heard fairly well. The facial paralysis was still pretty marked but was slowly passing off.

stage of illness

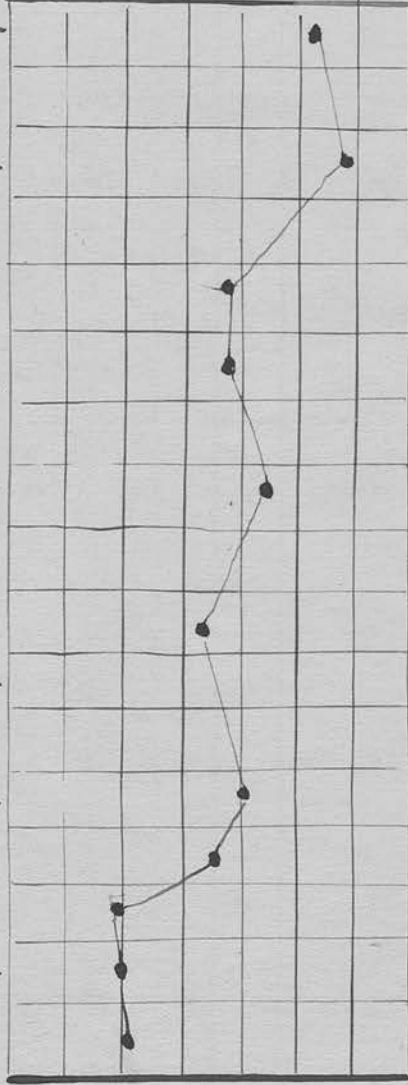


LEUCOCYTOSIS CHART.

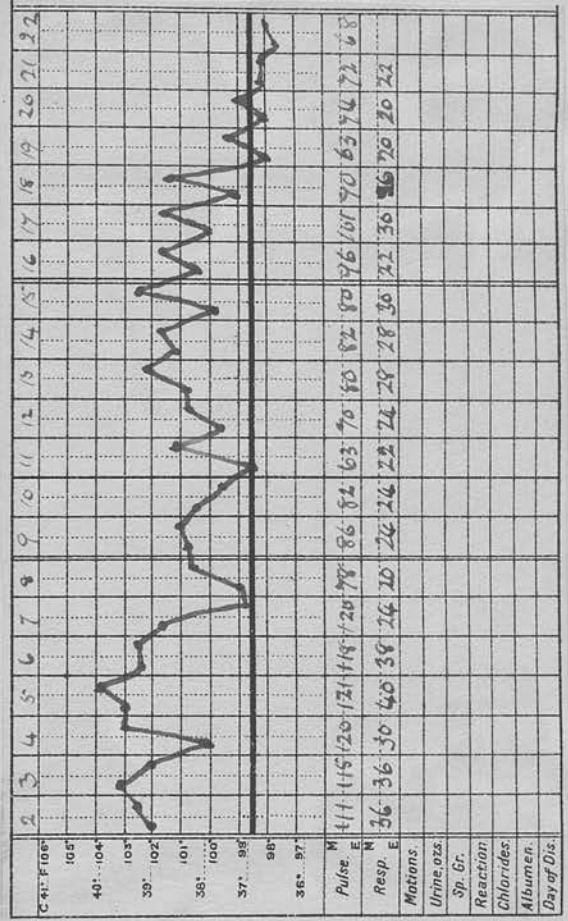
RECOVERY.

3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19

30 sec
1 min
1 1/2 min
2 min
2 1/2 min
3 min
3 1/2 min



COAGULATION CHART



XLI

TEMPERATURE CHART

	3	4	5	6	7	8	9	10	11	12	13	15	17
POLYMORPHONUCLEARS.	84.3	82	85.1	86.4	85.5	80.2	82	87.3	84	86	85	78	74
TRANSITIONAL	3	2.1	3	2	1	2	1.5	1.2	2	1	1.5	1	1
LARGE LYMPHOCYTES	6	6	5	6	4	12.1	9	7	8.1	7	4	5	4
SMALL LYMPHOCYTES	6.2	9.4	6	5.1	9.3	4	6.4	4.5	5	5	9	14.5	20
MYELOCYTES	-	-	.2	-	-	-	.2	-	-	-	-	-	-
EOSINOPHILE CELLS	.5	.5	.7	.5	.2	1.3	.8	.5	.9	1.	1.2	1.5	1.

1. Fork measure by no. lined.
2. Enlargement of do.

DIFFERENTIAL COUNT TABLE

CASE VII.

Man, Aged : 24

Occupation : Cabman

Residence : 11 Eglinton Street

Admitted : January 7th

Examined : do.

Complaint : Pain in right side of chest
with general weakness, cough
and sticky spit.

Duration : Five days.

History.

He lives a very exposed life and indulges considerably in alcohol, especially whisky. Comfortable home.

Previous Illness.

He says that since his childhood he has always been a healthy man.

Present Illness.

Five days ago he felt generally what he described as "seedy" and wretched when he was driving his cab, went home and took to his bed. He has no history of any distinct

rigor. The pain in his side came on and he got gradually worse until he was admitted.

State on Admission.

He is a moderately well developed man with slight cyanosis on his lips and some herpes. His face is pale and shows no malar flush.

Temperature 101 .6

Respiratory System.

His breathing is shallow and rapid 46 per min but regular and is costo abdominal. He has an intense stabbing pain in the right axillary region upon drawing a deep breath.

Cough, is short and sharp and comes in paroxysms and the spit brought up is fairly copious in amount and very tenacious. Rusty in color, and contains Fraenkels pneumococci.

Inspection.

Chest well formed and well clothed with muscle.

Expansion is less on the left side.

Palpation

Confirms this and shows that the vocal fremitus is less on the right side.

Percussion.

Absolute dullness over the lower and middle lobes of the right lung, otherwise the percussion is normal. Over this dull area loud bronchial breathing is heard with occasional bursts of medium crepitations, towards the end of inspiration.

In the axillary region on the right side medium coarse friction is heard.

Bronchophony is heard all over the dull area.

Alimentary System.

The usual phenomena in this disease the liver extends one inch below the costal margin in the mammary line.

Circulatory system.

The heart was normal and the pulse good but rapid 120 when admitted.

Nervous System.

He is very deaf in both ears and is very sleepless. There is no delirium and otherwise the nervous system is normal.

Urinary System.

Urine - straw colored, acid reaction, shows

diminution of Chlorides, specific gravity 1024.

No trace of Albumen.

Haemopoetic System.

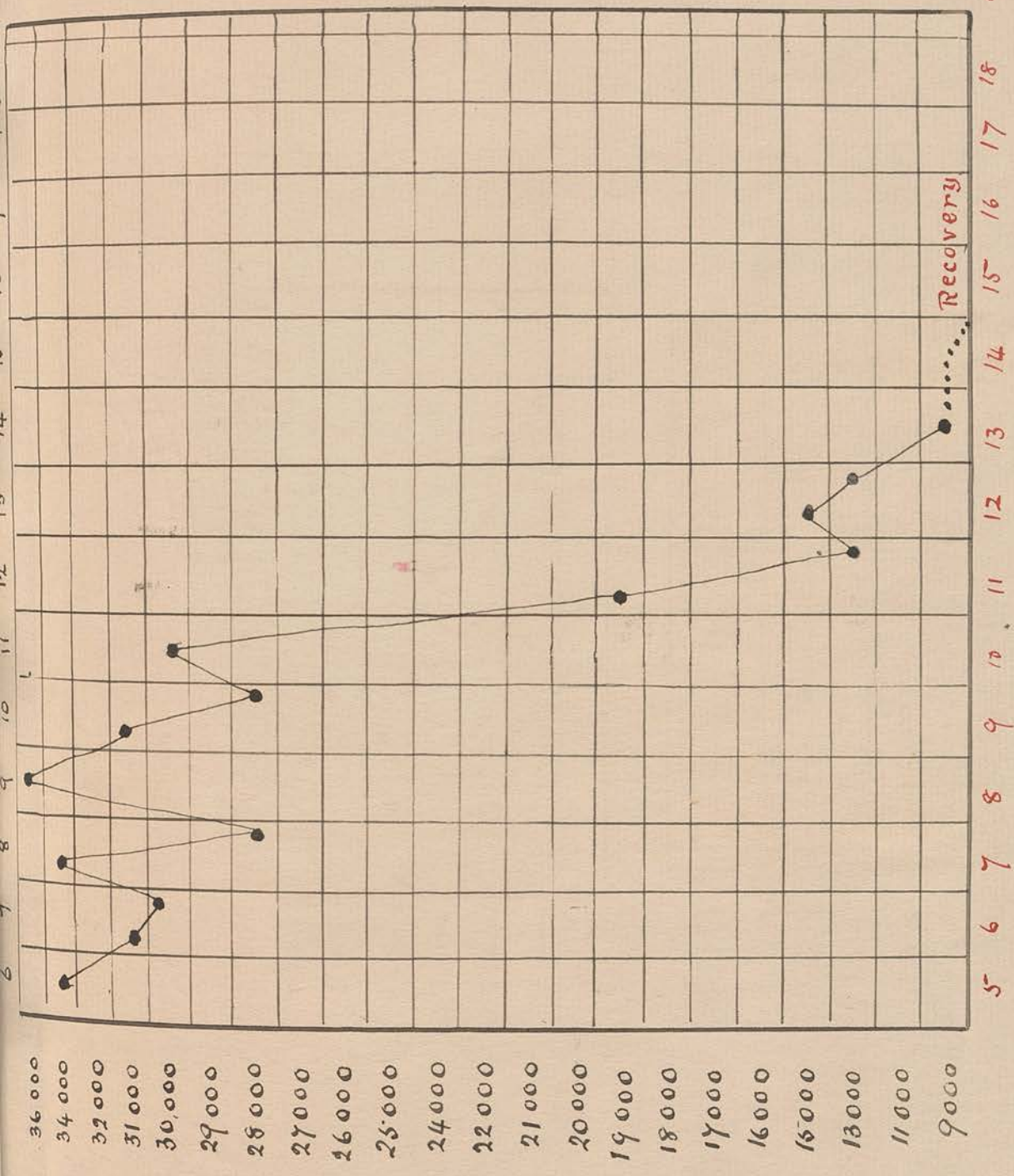
There is no enlargement of the spleen, thyroid or any glands.

Treatment.

Rest in bed, milk diet, Spr Ammon Aromat and Spr Aether Nit. On January 8th he was given oxygen to inhale and as his pulse was 134 and getting rather weak he was put on Liq Strychnina Hydrochlor and Tinct Strophanthus a.a. m V.

Progress.

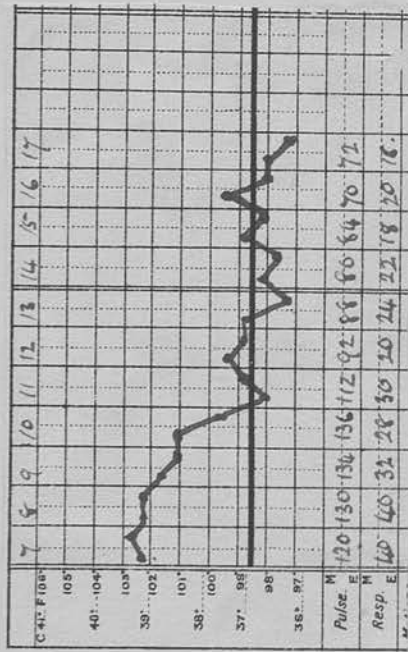
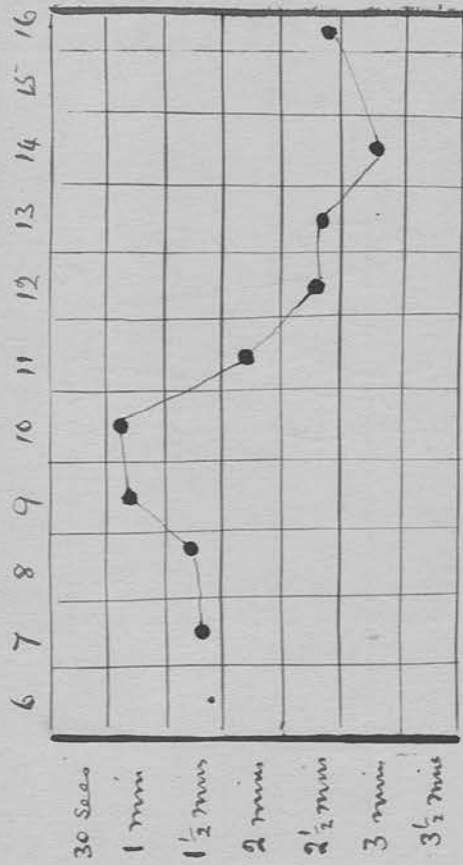
Up to the crisis he gradually got worse his respirations on the 9th January being 60 per min. and his pulse 134. On the 10th his temperature started to fall and reached normal. On the 11th, the physical signs gradually cleared off and he made a good recovery. Segments of tape worm were noticed in his stools. So on the 17th January he was given Felix Mas and castor oil and passed a large example of *Taenia mediocanellata* about 20 feet long. After this he rapidly progressed to recovery.



Recovery

LEUCOCYTOSIS CHART.

III



TEMPERATURE CHART

	6	9	12	16	20
POLYMORPHONUCLEARS	84.7	80	75	75	70
TRANSITIONALS	1	1.7	1.4	.8	.2
LARGE LYMPHOCYTES	6.3	7.6	14.3	10	8
SMALL LYMPHOCYTES	8	10	8	12	20
MYELOCYTES.	-	-	-	-	-
EOSINOPHILE	-	.7	1.3	2.2	1.8

DIFFERENTIAL COUNT TABLE.

CASE VIII.

Man, Age : 46

Occupation : Carter

Residence : Cowgate

Examined : ~~21st~~ 31st January

Family History : good

Personal History:

Is fairly comfortable at home but much exposed with his work. He occasionally has bursts of drinking but these do not often occur.

Previous Illnesses:

Always been a healthy man.

Present Illness:

This started two days ago. He had a shivering attack and went to bed with a severe pain on the right side of his chest. A cough developed and he got worse and was seen by me on the 31st January.

He was a well developed strong man with very good muscularity. He was slightly cyanosed but had no herpes.

Temperature 102.

Respiratory System.

His breathing was shallow and rapid 36 per min and regular, of costo abdominal type.

His cough was short and dry and he had no spit.

Physical Examination.

Showed a well formed chest well covered with muscle.

Inspection.

The left side moved more on respiration than the right. The vocal fremitus was somewhat increased on the right side.

Percussion.

Showed that the whole of the posterior surface of the right lung was dull.

There was some dullness at the left base.

Anteriorly the right lung had a somewhat high pitched note, the left was normal.

Auscultation.

Medium bronchial breathing over the right lung with some crepitations and increased vocal resonance.

Bronchial breathing also at left base with coarse crepitations and deep ronchi.

Circulatory System.

The heart is slightly enlarged and the sounds weak especially the first which is somewhat clicking. No murmurs are to be heard.

The Pulse

Is 104 regular in time and force, of medium tension, the wave was small and somewhat rapid in rise and fall.

Urinary System.

Shows no albumen or blood.

Nervous System.

He is inclined to be delirious and is somewhat restless and sleepless.

Integumentary System.

He is perspiring freely the skin being quite moist. No herpes nor any eruption on his body.

Haemopoetic System.

Normal.

Alimentary System.

Tongue dry and lips cracked with sordes on the teeth. His bowels are very confined.

Treatment.

He was given Spr Ammon Aromat. m 30 Liq Ammon Acet every four hours. He was also given Tinct. Digitalis m 10 every four hours and ordered whisky, Kept in bed milk diet. He was ordered to be cold sponged when he felt uncomfortably warm.

Progress.

For the next two days he held his own, his temperature varying from 101 to 103. His pulse however was getting more rapid and weaker. The physical signs remained unchanged. On the following day, he was worse, his pulse being very weak, and so rapid, it could hardly be counted, and he died on this the sixth day of his illness.

No Post Mortem was obtained.

8 day of illness

2

7

6

5

4

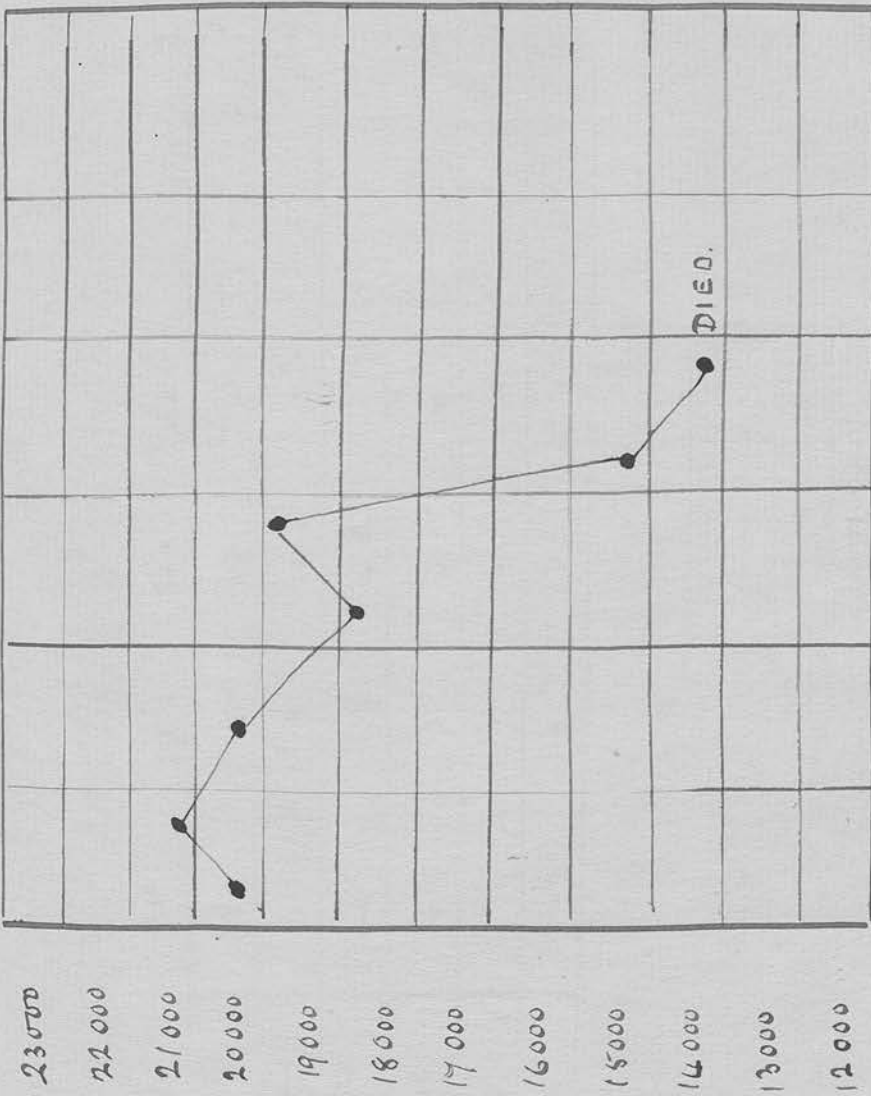
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28

29

30

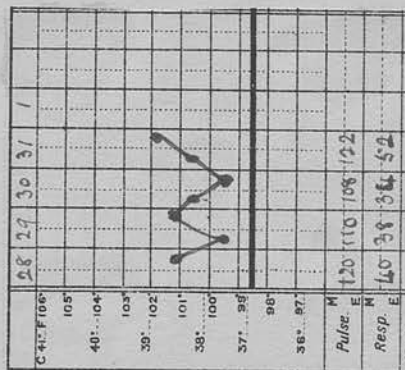
31



LEUCOCYTOSIS TABLE.

	28	29	30	31	1	2
POLY MORPHONUCLEARS	78	79.5	82	90.1	-	-
TRANSITIONAL CELLS	1	1.3	1.2	1.3	-	-
LARGE LYMPHOCYTES	14	11.2	6.8	5.4	-	-
SMALL LYMPHOCYTES	6.8	8	10	4.2	-	-
MYELOCYTES	-	-	-	-	-	-
EUSINOPHILES	.2	-	-	-	-	-

DIFFERENTIAL COUNT TABLE



TEMPERATURE CHART.

CASE IX.

Man Aged : 34

Occupation : labourer

Admitted : January 2nd.

Examined : do.

Complains of shortness of breath and
general malaise.

Duration : three days.

History:

He is an only child, his father and mother are both dead the causes are unknown.

He has always had plenty of good food, but often with long fasts between. He is moderate in his use of alcohol, but is a heavy smoker, consuming one pound of tobacco a week. His home is in a lodging house along with eighteen other lodgers, but comfortable.

Previous Illnesses.

Does not remember any, except pains in his legs.

Present Illness.

For the last five weeks he has been catching

chill on chill till on Saturday December 28th he took one rather worse than usual. He went to the Cowgate Dispensary and was sent in here.

On Admission.

He is a strong hardy looking man with good muscularity. There is slight cyanosis but no herpes.

Temperature 102 .5

Respiratory System.

He has a well formed chest but showing some slight hollowing under the left clavicle.

Both sides, moved equally on respiration, and at this date no physical signs could be made out except some emphysema of both lungs. But Two days later the following appeared.-

Breathing 26 per min is costo abdominal and causes him no pain. He has a short cough and very sticky sputum, more of a yellow color than rusty. It is muco purulent and shows many pneumococci.

There is now marked dullness over the middle lobe of the right lung with medium bronchial breathing. Above this, there are

some crepitations. Vocal Resonance is increased over this area.

Circulatory System.

This shows very little, the heart being evidently normal and the pulse fairly good, regular, and 104 in rate. The artery wall was a little thickened.

Alimentary System.

He is very much inclined to vomit after anything that he takes. Otherwise there is the usual loss of appetite etc. found in this disease.

Haemopoetic System.

There are a few enlarged glands in his groins otherwise normal.

Nervous System.

Great sleeplessness but no delirium.

Urinary System.

Urine color dark amber specific gravity 1023. The reaction acid there are traces of albumen and a deposit of mucus.

Treatment.

He was given Pot Iodid gr V Spr Ammon Aromat

and Tinct Camph Co a a M 20 and on the 3rd of January Tr Strophanthus M V every four hours was added.

Progress.

His temperature kept about 102 until the 5th of January and fell slowly with the pulse, until it reached normal on the 8th. There was some diminution of the area of dullness and the crepitations were somewhat coarse. On the 10th of January the temperature was rising, and there was a good deal of dullness over the right lower lobe medium friction was also well marked. His pulse remained satisfactory being only 80 although the temperature was 102.

After this date, his respirations became much more rapid rising to 52 per min on the 13th. He was given oxygen to inhale. A great deal of cyanosis was now present, and his pulse though only 108 was very weak and dichrotic. He was somewhat delirious and died during the early morning of the 14th of January.

Post Mortem Report.

He is a moderately well developed man whose rigidity is passing off and there is a lot of lividity.

The pericardium contains two ozs. of serum.

The left Pleura contains 2 oz. of fluid.

The right Pleura is bound down by dense old adhesions.

The Heart shows some fatty change. The right auricle and left auricle are slightly dilated and none of the chambers contain antemortem clots.

Left Lung: weight 13 oz. It is emphysematous and shows no consolidation.

The Bronchi contain muco pus. The Bronchial glands are pigmented and contain some old tubercular foci.

Right Lung: weight 2 lbs. 13 oz. The visceral pleura shows some recent pleurisy.

The Bronchi are congested and contain muco pus and some haemorrhages.

The Bronchial glands are pigmented, no caseation and the pulmonary vessels are empty.

Lung Tissue.

Upper lobe - The pleura is adherent and the

lung emphysematous. There is a collection of some yellow pus between the upper and middle lobes.

Middle lobe - is congested and shows some broncho pneumonia inferiorly. *Part* of which shows a tendency to necrosis. Purulent points are seen through out.

Lower lobe - This shows diffuse consolidation and the appearance of confluent Broncho pneumonia. On pressure a considerable amount of purulent matter is squeezed out.

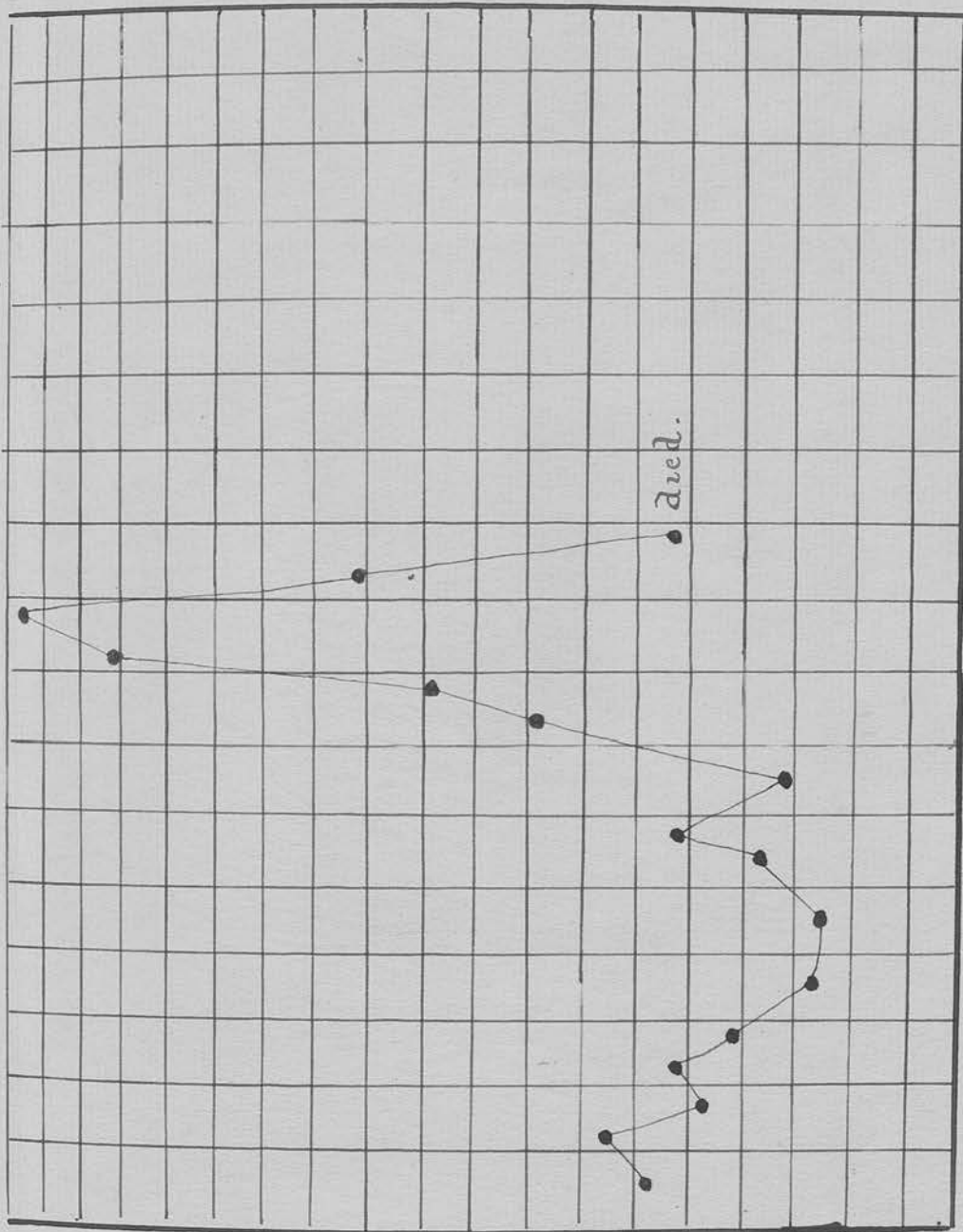
Liver shows cloudy swelling.

Spleen : 3 oz. is small and firm on section with minute haemorrhages.

Kidneys show some congestion and the capsules thick and adherent.

4 5 6 7 8 9 10 11 12 13 14 day of month

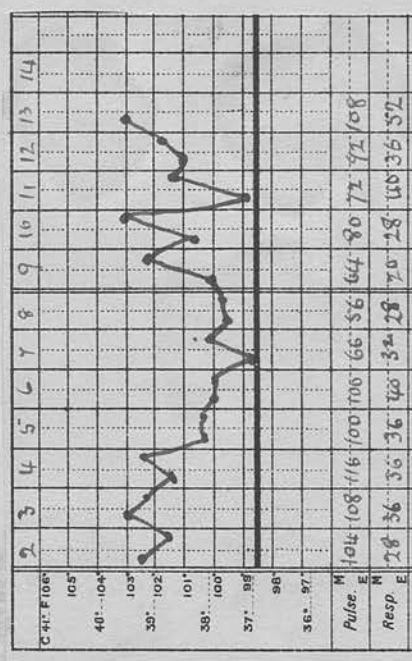
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died.

LEUCOCYTOSIS CHART.

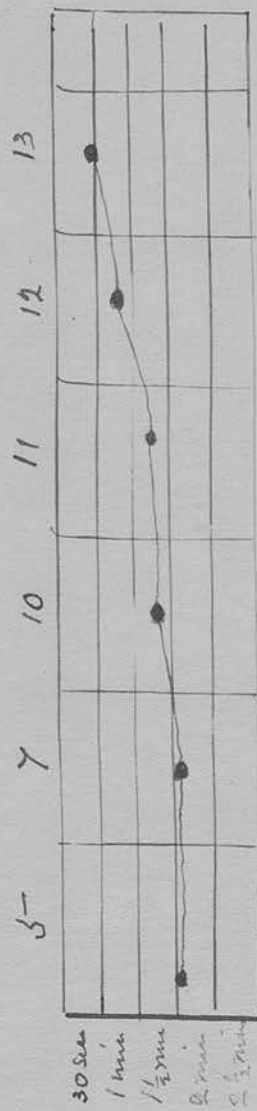
3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 day of month



TEMPERATURE CHART

	5-	7	9	11	12	13.
POLY MORPHO NUCLEARS	90.2	91	90.	88	89	81
TRANSITIONAL CELLS	1	.8	2.	1	2	2
LARGE LYMPHOCYTES	4.3	5	5	6.3	4.3	6
SMALL LYMPHOCYTES	4	3	3	4	4	8
MYELOCYTES	.5	.2	.	.7	.5	3
EOSINOPHILES.	-	-	-	-	.2	-

DIFFERENTIAL COUNT TABLE



COAGULATION TABLE

CASE X.

Man Aged : 30

Occupation : Varman

Residence : Leith Street

Admitted : January 17th

Complaint : Cough and pain in the left
side.

Duration : Five days.

Family History.

Only child both parents died in old age.

Father of an accident.

Personal.

Comfortable home in lodgings with plenty of good food. He has exceeded somewhat in alcohol for the last four years. His work is hard twelve hours and exposed to all weathers.

Previous Illnesses.

He was ill for a fortnight with Rheumatic fever when seventeen years old but quite recovered.

Present Illness.

No history of a rigor but the illness commenced on Monday with pain in his left side. He went to bed was somewhat better for the first day or two and then got worse till admission.

State on Admission.

Patient is a poorly developed spare man no herpes or cyanosis.

Temperature 100 .4

Respiratory System.

Respirations 32 per min with much pain in left side. His cough is not very troublesome. Spit : this is frothy muco pus with many Fraenkels diplococci and is not very tenacious. No tubercle bacilli could be found. His chest is not well clothed with muscle and shows hollowing under both clavicles. The right side moves more freely than the left. Vocal fremitus is about equal on the two sides.

Percussion.

Shows involvement of the whole left lower lobe and also of the left upper lobe where the dullness is more marked than at the base.

at the left base the breathing is broncho vesicular and medium pitched bronchial at the apex - Medium crepitations are heard all over the lung especially during inspiration. Anteriorly well marked friction is heard over the painful area of the left lung. Right Lung normal.

Alimentary System.

There is nothing abnormal except loss of appetite etc. The liver is slightly enlarged and extends half an inch below the costal margin in the mammary line.

Circulatory system.

The heart is not enlarged. There is a slight presystolic murmur in the mitral region. Otherwise normal. Pulse is good medium tension regular 104 in rate and the vessel wall a little thickened.

Urinary System.

Urine color buff reaction acid specific gravity 1018. A deposit of urates, a trace of albumen and the Chlorides diminished.

Nervous System.

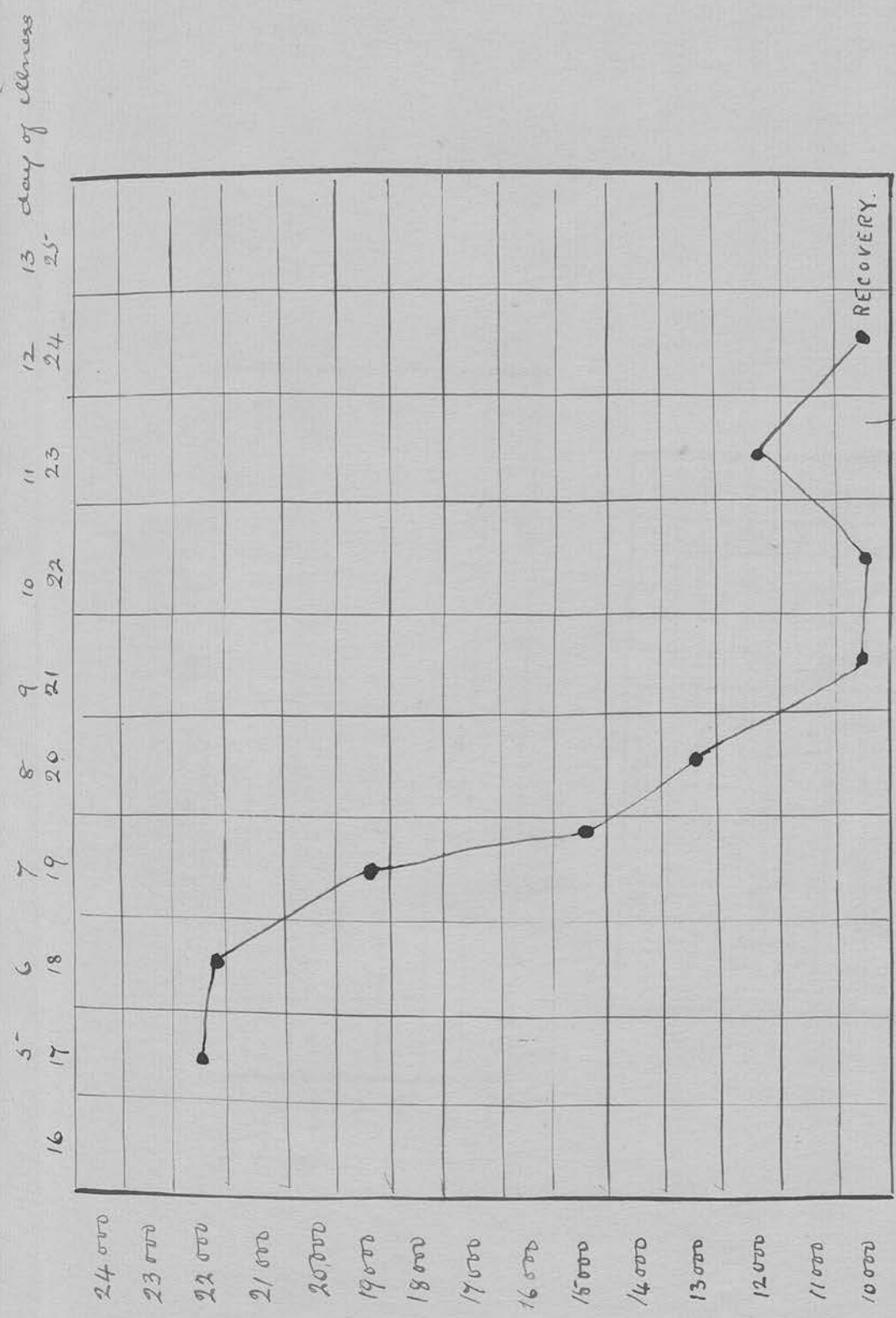
Does not sleep well but there is no delirium.

Treatment.

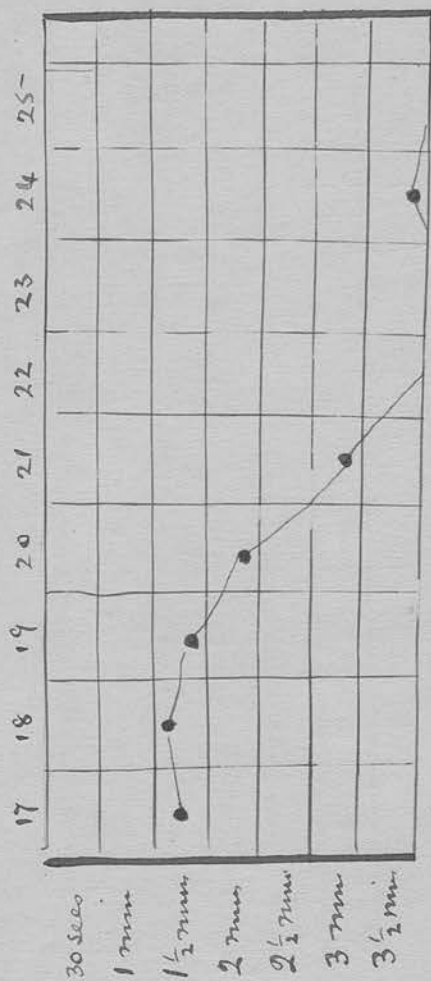
Rest and milk diet with Pot Iodid gr V Spr
Ammon Aromat M 30 every four hours, Ice bag
was applied for the pain in the side.

Progress.

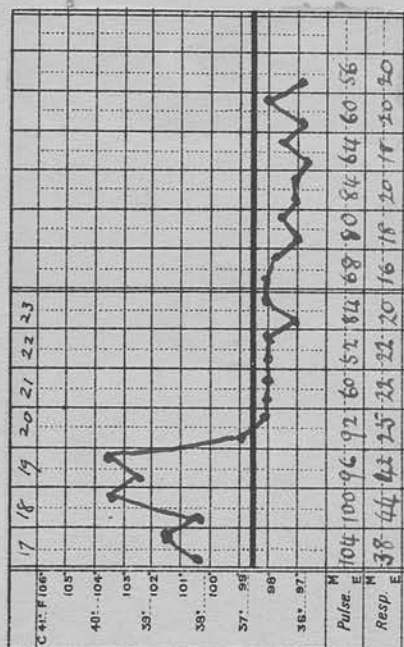
The day after admission he was given Tinct
Strophanthus M V. every two hours as his
pulse was somewhat weaker. On this day
the albumen had disappeared and the dullness
was clearing from the base. The crepitations
were more marked. On the night of the 19th
Patient had his crisis the temperature drop -
:ping from 103 .5 to normal. After this he
progressed favorably. The dullness cleared
off the left base. Crepitations were more
plentiful and the friction was very coarse.
Later the dullness also cleared from the apex
and he was dismissed cured.



LEUCOCYTOSIS CHART.



COAGULATION CHART



TEMPERATURE CHART

	17	18	19	20	22	25-
POLYMORPHONUCLEAR CELLS.	80.5	81.2	79	75.8	72	70.1
TRANSITIONAL CELLS	2	1.2	1	1	1.3	-
LARGE LYMPHOCYTES.	10.3	12	10	9.2	8	6.1
SMALL LYMPHOCYTES	5	4.2	9	13	16.7	22.2
MYELOCYTES	2	1	.5-	-	-	-
EOSINOPHILES.	.2	.4	.5-	1	2	1.6

DIFFERENTIAL COUNT TABLE.

CASE XI.

Man Aged : 46

 Occupation : labourer

 Residence : Penicuik

 Admitted : January 30th

 Examined : do.

 Complaint : severe pain in left side
 with weakness, restlessness
 cough and spit.

 Duration : Three days.

History, Family

Satisfactory.

Food and Drink.

As a rule is well fed and does not drink alcohol to any excess.

Surroundings.

He is a labourer and works at the Talla water works. Three weeks ago he had an attack of pneumonia, so the Doctor called it, and he was in bed for four days. He came down on being convalescent from Talla to Penicuik and

regained his strength so rapidly that he was able to return to work for three days. Then two days before admission he was taken bad again with fever and pain in his left side. He got worse and was sent in to the Edinburgh Royal Infirmary.

State on Admission.

Patient is a strong well developed man. He shows no herpes.

Temperature 101 .2

Respiratory System.

He is lying on his left side with rapid regular respirations 50 per min costo abdominal in type. He has a somewhat severe cough causing him great pain.

Spit: Thick and sticky muco purulent with the majority of the cells present polymorphonuclear cells in more or less broken down condition. The spit is crowded with Fraenkels pneumococci. The spit is somewhat rusty in color.

Physical Signs.-

The Left Lung : The upper lobe is somewhat tympanitic and the breathing broncho vesicular

regained his strength so rapidly that he was able to return to work for three days. Then two days before admission he was taken bad again with fever and pain in his left side. He got worse and was sent in to the Edinburgh Royal Infirmary.

State on Admission.

Patient is a strong well developed man. He shows no herpes.

Temperature 101 .2

Respiratory System.

He is lying on his left side with rapid regular respirations 50 per min costo abdominal in type. He has a somewhat severe cough causing him great pain.

Spit: Thick and sticky muco purulent with the majority of the cells present polymorphonuclear cells in more or less broken down condition.

The spit is crowded with Fraenkels pneumococci.

The spit is somewhat rusty in color.

Physical Signs.-

The Left Lung : The upper lobe is somewhat tympanitic and the breathing broncho vesicular

with medium crepitations and ronchi.

The lower lobe shows marked dullness all over its area with loud medium pitched bronchial breathing and increased Vocal Resonance a few fine crepitations could be made out. Slight friction is heard in the left axillary region.

The Right Lung expands better than the left and on percussion is normal on auscultation at the base the breathing is broncho vesicular and there are numerous ronchi. Over the rest of the lung the breath sounds are harsh.

Circulatory System.

The heart is slightly enlarged, the apex being in the sixth Interspace. The sounds are closed except in the aortic region, where a faint double aortic murmur can be made out. The pulse is regular in time and force with a full wave and low tension. Rapidity 140 Artery wall a trifle thickened.

Urinary System.

Urine color amber Specific gravity 1026 reaction acid. A trace of albumen is present.

Haemopoetic System.

No enlarged glands. The spleen is slightly enlarged. The other systems show nothing abnormal.

Progress and Treatment.

The Patient was given Pot Iodid gr V. Spr Ammon Aromat Tr Camph Co a a M 20 also Tinct Strophanthus M V. Whisky every four hours. The patient's condition gradually got worse. His breathing and pulse more rapid and weaker. The Physical signs remained unchanged and he died at 6.15 p.m. on February 1st.

Post Mortem Report.

Strong well developed man with slight lividity on the thorax and neck.

The Pericardium contains of straw colored fluid with flakes of lymph.

Both Pleurae contain old adhesions over both lobes.

Heart : weight 15 oz.

general appearance : The auricles are distended and there is a large antemortem clot in the

pulmonary artery. The aortic valve is not competent. The orifice is blocked with clot. The segments are thickened from old endocarditis and adherent up to the corpus aurantii.

The coronary arteries show slight atheroma.

Left Lung weighs 5 lbs, 1 oz.

The visceral pleura is thickened and adherent to the parietal.

The Bronchi show Bronchitis.

Lung Tissue.

Upper lobe congested and oedematous and consolidated at its lower part. This is the stage of Red Hepatization.

Lower lobe consolidated completely and grey purulent matter can be scraped out. It is haemorrhagic at the base, and shows beginning necrosis and breaking down.

Right Lung weighs 1 lb. 12 oz.

Bronchi congested

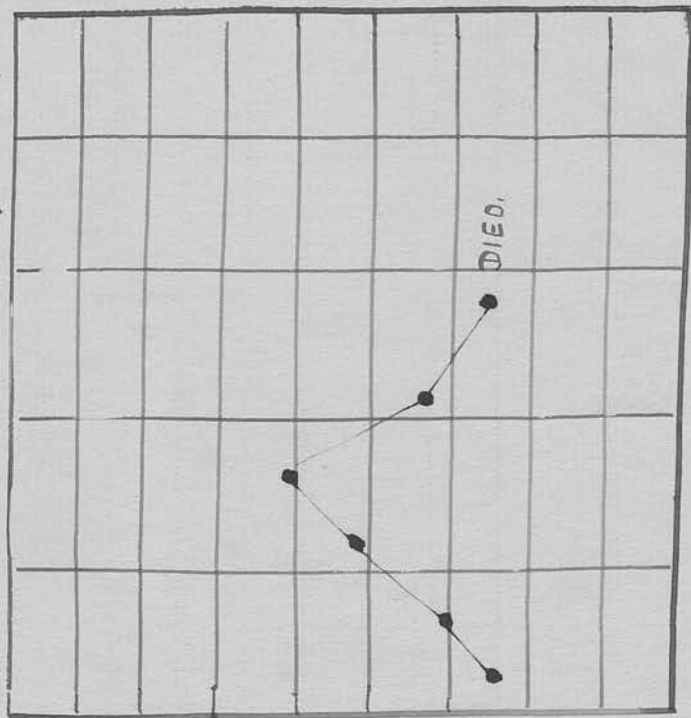
Lung Tissue.

Upper lobe oedematous

Lower lobe is friable.

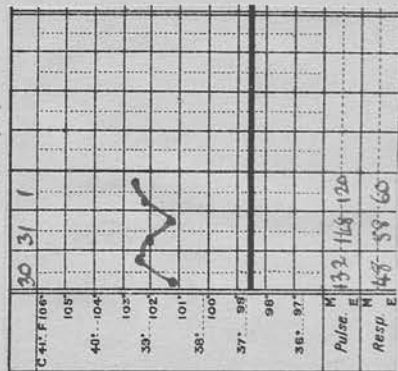
The spleen weighs 9 oz. and is semi-diffuent and shows small haemorrhages.

3 4 5 6 7 day of illness
30 31 1 2 3

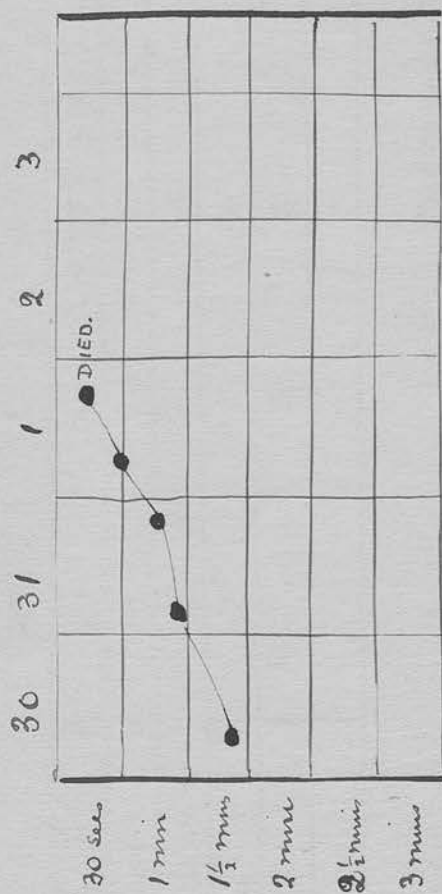


15000
14000
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9000
8000
7000

LEUCOCYTOSIS CHART



TEMPERATURE CHART



COAGULATION CHART.

30 31 1

POLYMORPHONUCLEARS	80	72	78
TRANSITIONALS	7.2	-	8
LARGE LYMPHOCYTES	5.	-	6
SMALL DO.	7.	-	6
MYELOCYTES	.8	-	2.
EOSINOPHILES	-	-	-

DIFFERENTIAL COUNT TABLE

BILOBED.
HORSE SHOE
MULTILOBED.

1

16
54
30

SHAPE OF NUCLEUS.

CASE XII.

Man Aged : 22

Occupation : baker

Residence : 73 Albert Street

Admitted : February 10th

Examined : do.

Complaint : Cough and spit with pain in
left side of chest.

Duration : Six days.

History.

No history of tubercular mischief in any other member of the family.

He is regular in his habits, gets well fed and is very temperate in alcohol.

At work in a bakehouse, he is naturally much exposed.

Previous Illnesses.

His mother says as a boy he was somewhat weak and often had attacks of bronchitis. For the last year he has been rather troubled with a severe cough, which is worse in the morning and he had a copious spit. He has been

falling off in weight.

Present Illness.

He was quite well when he went to bed on the night of the 4th but in the morning he had a rigor and severe pain in the left side. He kept his bed and has got gradually worse.

The last two nights he has been very delirious and so was sent in on the 10th of February.

State on Admission.

He is a thin spare man with poor muscularity. His cheeks are rather shrunken and he looks moderately cyanosed. No herpes present.

Temperature 103.

Respiratory system.

Patient is lying on his back and breathing 36 per min and somewhat shallow. He has a short cough and his spit is tenacious, yellow in color, rather frothy, large in quantity, mucus purulent contains a few tubercle bacilli and is crowded with Fraenkels pneumococci.

He has a severe pain in the left side reaching round into the small of his back.

Inspection.

The Right Lung moves more freely on respiration than the left. The left side of the chest is red and skinned with poultices. The chest is flattened at the left apex, and shows supra and infra clavicular hollowing.

Palpation.

Vocal fremitus is increased on right side.

Percussion.

On admission the whole of the left upper lobe was dull, both anteriorly and posteriorly. Two days later the left lower lobe also became dull. The upper lobe had medium bronchial breathing and crepitations markedly consonating also coarse friction anteriorly and at the lower margin of this lobe. The lower lobe to commence with, showed nothing, except harsh breath sounds, and prolonged expiration but after two days, when the dullness was marked bronchial breathing was found.

The Right Lung was normal, except slight impairment of the note at the apex of the lung.

Circulatory System.

The heart is normal in size and the sounds closed. There seems slight accentuation

of the Pulmonary Second sound. Pulse 120, regular in time, and force, and rather low tension and poor volume.

Nervous System.

Delirium at night otherwise normal. The other systems only showed the changes customary in Pneumonia.

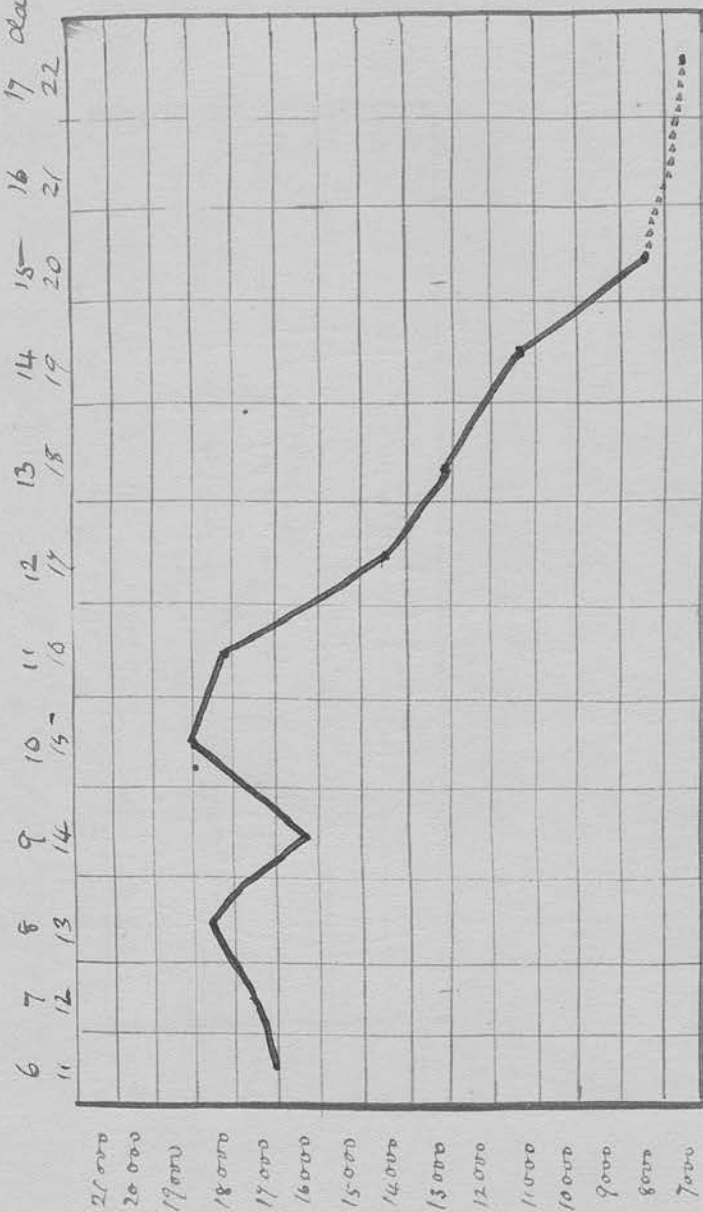
Progress and Treatment.

He got Pot Iodid gr V Spr Ammon Aromat m 30 every four hours. Tinct Strophanthus m V every four hours was given later.

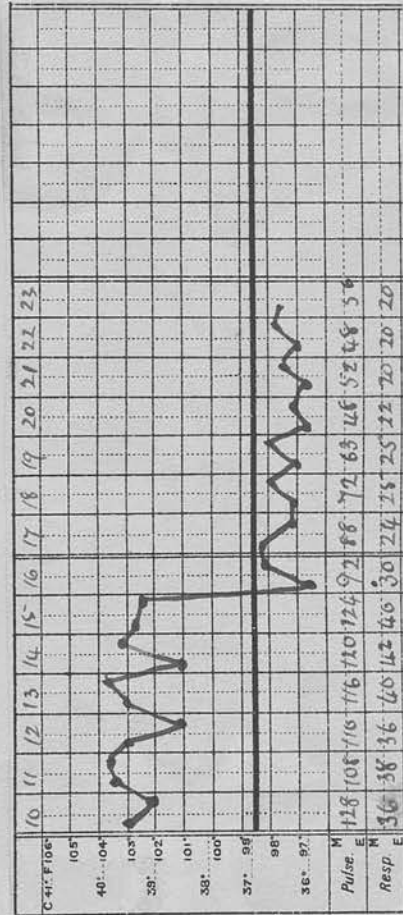
On the 15th he had his crisis that is on 12th day of his illness. His temperature fell from 103 to normal, and his pulse from 120 to 92. After this, he progressed steadily. The dullness and physical signs cleared off well from the left base. A good deal of dullness remained at the left apex. He rapidly regained strength and was discharged from the ward on February 30th.

I must now condense my remaining cases somewhat more. They are all cases showing little abnormal about

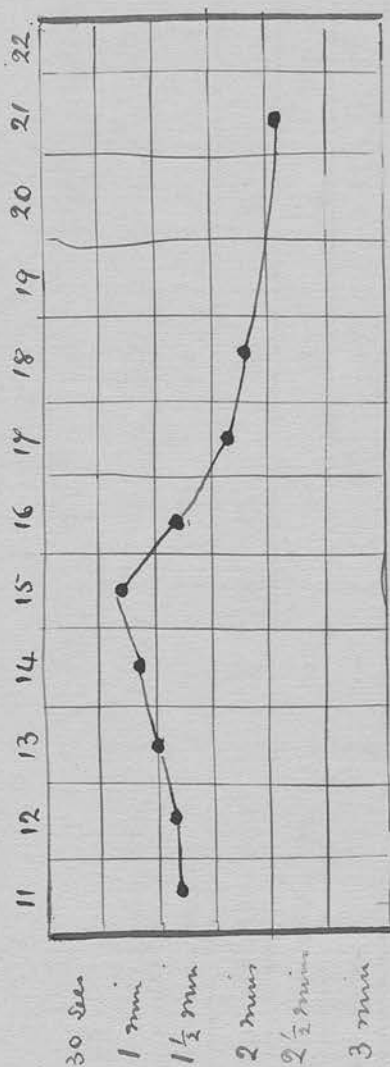
17 day of illness



LEUCOCYTOSIS CHART



TEMPERATURE CHART



COAGULATION CHART.

	11	13	15	17	23.
POLYMORPHONUCLEARS	90.7	88	90	92	86
TRANSITIONALS	1	2	1	1	.5-
LARGE LYMPHOCYTES	2	3	4	2	2
SMALL LYMPHOCYTES	6.1	6	4	4	10
MYELOCYTES	-	.5-	.7	.5-	.5
EOSINOPHILES	.2	.5	.3.	.5-	1.5.

DIFFERENTIAL COUNT TABLE

them. The first two are fatal cases, the rest
going to a satisfactory termination.

CASE XIII.

Man Aged : 62

Occupation : Stableman

Admitted : March 13th

Complaint : pain in left side of chest,
with cough and spit.

Duration : One week.

History, Family : good

Personal : Surroundings satisfactory.

Temperate with alcohol, and has
always been a healthy man.

Present Illness:

Commenced with rigor, and then the symptoms
of a left sided pneumonia came on, and he
was sent in on March 13th.

State on Admission.

Thin man with fair muscularity for his years.
slight cyanosis, and his temporal arteries
stand out markedly and are tortuous.
He showed a left lower lobe pneumonia, and
bronchitis. The Lungs otherwise were
emphysematous.

Circulatory System.

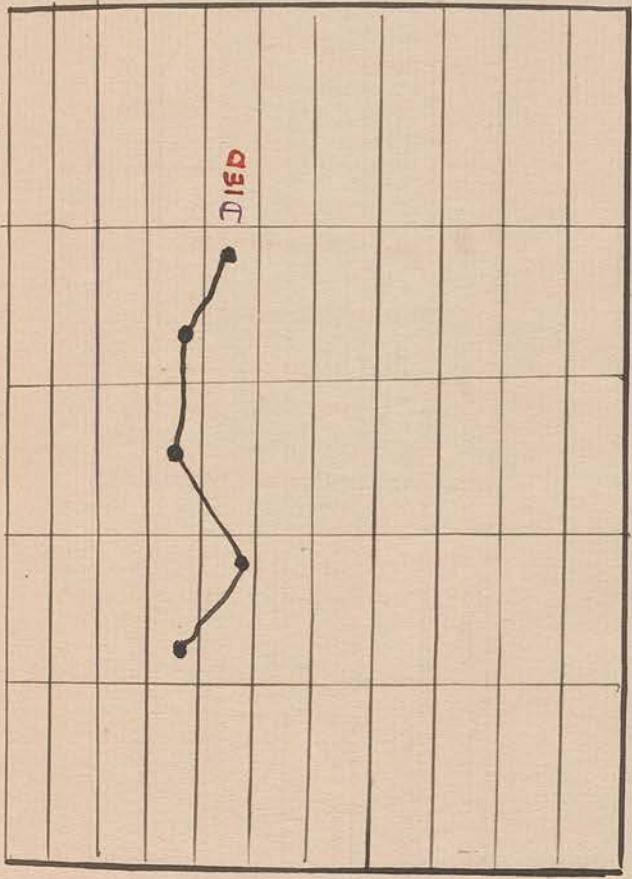
Showed nothing marked, except an accentuated aortic second sound, and great thickening and tortuosity of the peripheral arteries. The other systems showed nothing abnormal to the disease.

Progress.

He gradually became weaker. His pulse rapid and weak. He was unable to spit up the sticky spit and he died in the early morning of the 15th.

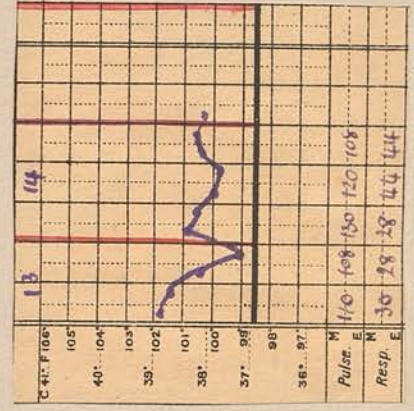
No Post Mortem was obtained.

7 8 9 10 11 day of illness
12 13 14 15 16



DIED

LEUCOCYTOSIS CHART



TEMPERATURE CHART

XL

Polymorphonuclear cells	92	90	90
Transitional cells	4	2	2.3
Large Lymphocytes	2	4	3
Small Lymphocytes	1.5	3	2.5
Myelocytes	1.5	1	2.2
Eosinophil cells	#	#	-

DIFFERENTIAL COUNT TABLE

CASE XIV.

Man Aged : 19

Occupation : Brewer

Examined : February 23rd

History.

His family history is satisfactory and his home comfortable. He is a good deal exposed at his work. No history of alcoholic tendencies.

Present Illness.

This started with a rigor and pains all over his body, and he got steadily worse.

Duration: Three days.

State on examination.

He is a well developed lad, showing herpes on lips, no other obvious morbid tendencies.

TemperatureRespiratory System.

Breathing 32 per min, and very shallow.

Short cough and very tenacious spit, small in quantity and rusty in color showing

pneumococci.

Physical Examination.

Showed pneumonia of right lower and middle lobes. The right upper lobe was somewhat tympanitic. Some friction was heard in the right axillary region.

Circulatory System.

Slight Presystolic in the mitral area and reduplication of the second sound, otherwise normal. Pulse Rate 102 regular in time and force and of rather low tension.

Other symptoms - Slight delirium at night.

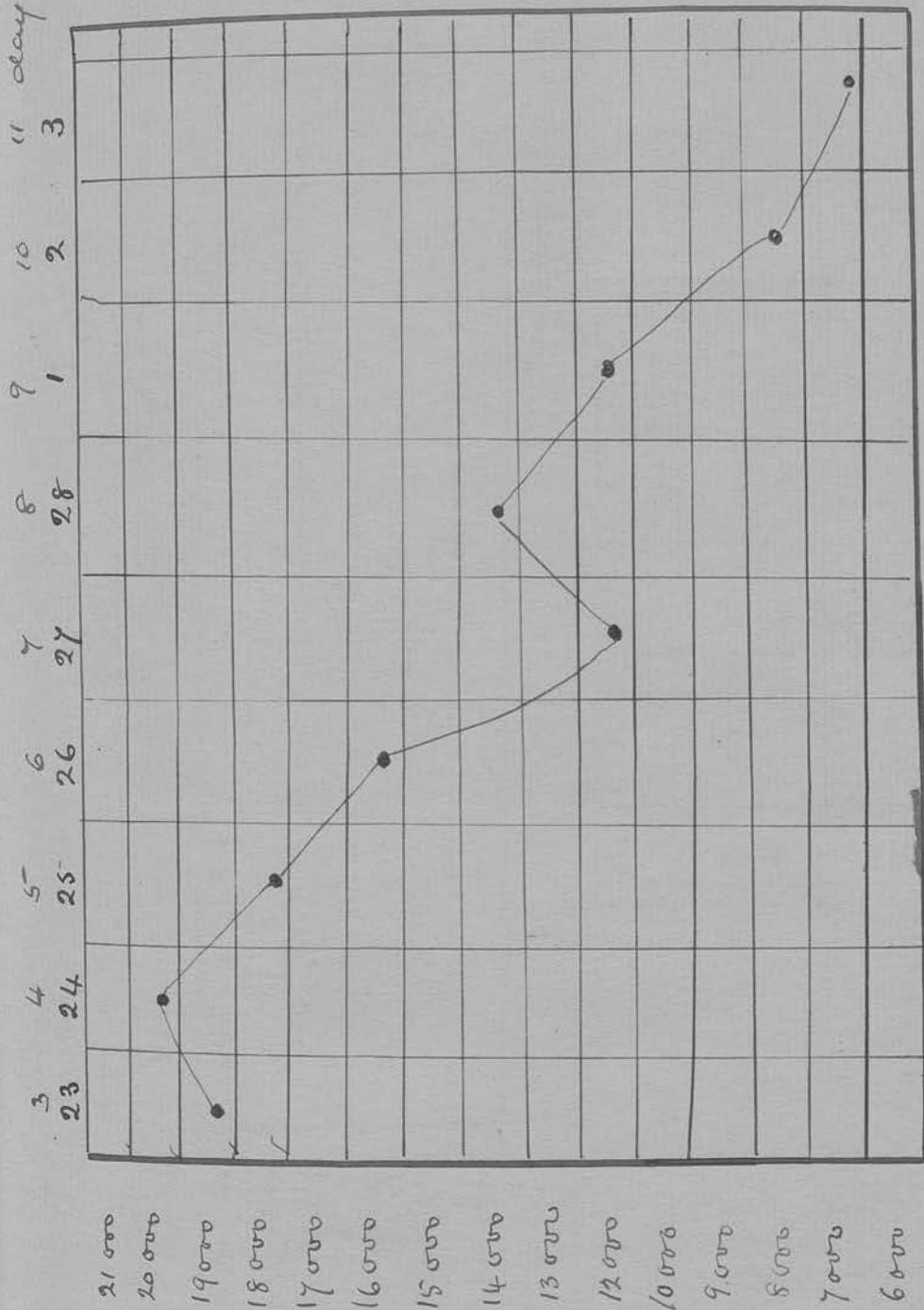
- Poor appetite, etc.

Progress.

On the fifth day he had his crisis the temperature falling from 102 to normal.

After this the Physical signs cleared rapidly off.

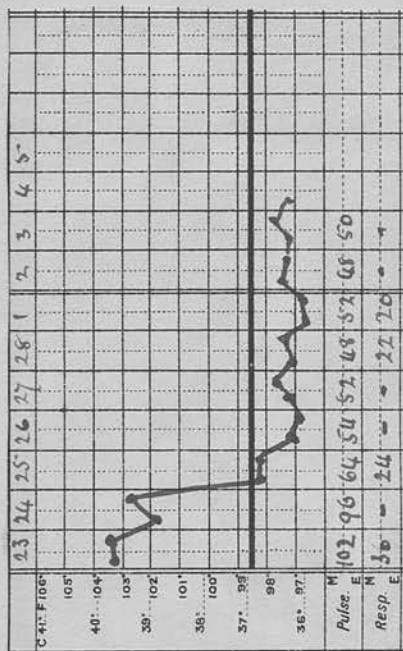
" day of illness



LEUCOCYTOSIS CHART.

DIFFERENTIAL COUNT TABLE

	23	24	26	1
POLYMORPHS.	90.1	84.8	84	78.
TRANSITIONALS	2	1	2	.8
LARGE LYMPHS	3.9	6.2	11	6
SMALL DO	4	8	3	14.1
MYELOCYTES	-	-	-	-
EOSINOPHILES	-	-	-	1.1



TEMPERATURE CHART

CASE XV.

Man Age : 48
 Occupation : Blacksmith
 Examined : February 25th

History.

He has always led a steady life, taken little alcohol. His home is comfortable but he is a good deal exposed at his work. He has always been a healthy man.

Present Illness.

He was seized with a rigor while engaged in his work four days ago, he went home and took to his bed feeling a severe pain in his side. He gradually got worse and I saw him on the 25th.

State on Examination.

Very strong well developed man, large chest
no cyanosis, nor herpes.

Temperature 101 .4

Respiratory System.

Respirations 36 per min. Cough apt to come

in paroxysms, and his spit is very tenacious, of a pale yellow color and with streaks of blood in it. It contains pneumococci. Consolidation of the right lower lobe was found with bronchial breathing, and increased Vocal Resonance. Otherwise the lungs were normal.

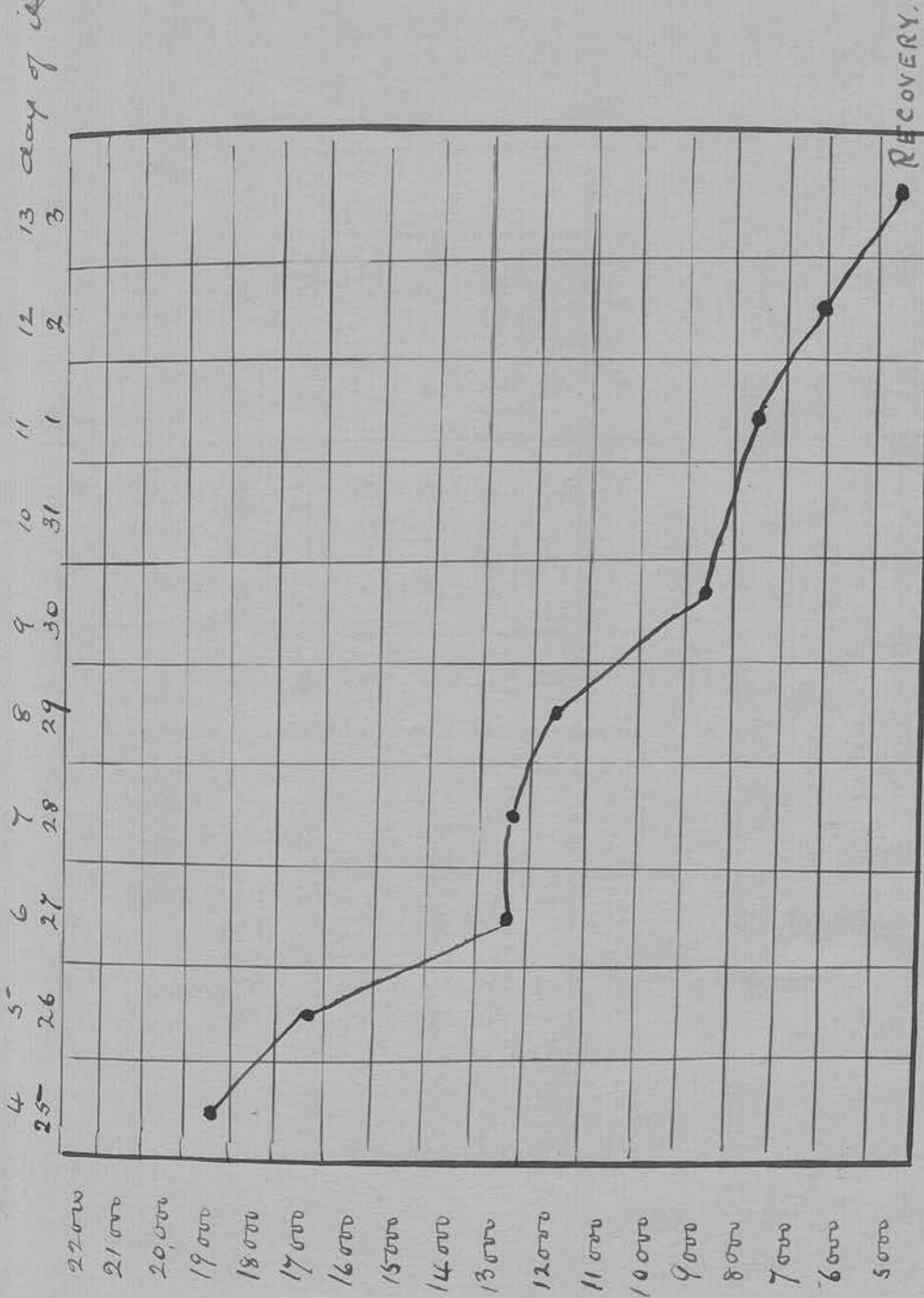
Circulatory System.

The apex beat was just external to nipple line. A slight blowing systolic was heard in the mitral area, conducted to the axilla and the Pulmonary second sound was accentuated. Pulse 120, moderate tension and volume, no thickening of artery wall. Other systems show nothing abnormal.

Progress.

He had no marked crisis, the temperature reached normal on the 9th day and he progressed favorably to recovery.

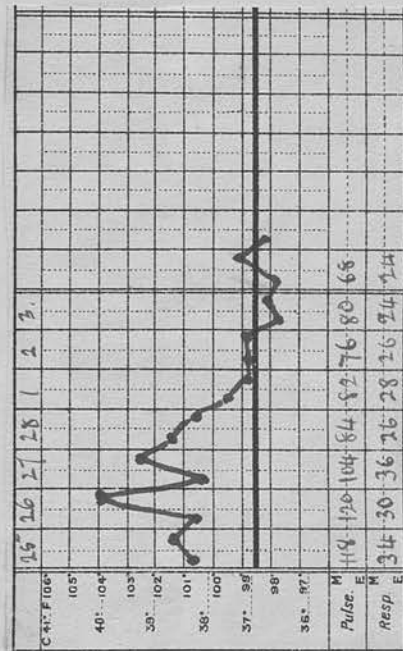
13 day of illness.



LEUCOCYTOSIS CHART.

1	25	26	29	31	3
Polymorphonuclear cells	91	90	82	74	65.4
Transitional cells	.5	1	1	1.5	.2
Large Lymphocytes	6	5	7	6	5.4
Small Lymphocytes	2	3.5	9	18	26
Myelocytes	.5	.5	.7	-	
Eosinophile cells.	-	-	.2	.5	3.

DIFFERENTIAL COUNT TABLE



TEMPERATURE CHART.

CASE XVI.

Man Age : 19
Occupation : Brassfinisher
Admitted : March 14th
Examined : do.
Complaint : pain in side
Duration : three days.

History.

Patient has comfortable home and is well looked after. He is well fed and takes little alcohol. Previously Healthy.

Present Illness.

This started three days ago with shivering pain in the right side and cough.

State on Admission.

Well developed lad with herpes on lips otherwise no morbid appearances.

Temperature 103.

Respiratory System.

Respirations 50 per min shallow and painful
Cough short and does not bother him very much.

Spit tenacious, rusty and contains pneumococci

Physical Examination.

Shows great loss of movement on the right side. Entire dullness of the whole of the right lung and marked dullness over the left base to the angle of the scapula. The remainder seems somewhat tympanitic. This dull area shows bronchial breathing, fine crepitations, and diminished vocal resonance at the right base, elsewhere it is increased.

Circulatory System.

The heart is normal in size, and the sounds closed. The pulse is 130 regular in time and force, low tension with a small wave. The vessel wall is not thickened.

Other symptoms show nothing abnormal to an attack of pneumonia. He is slightly delirious

Urine contains a trace of albumen.

Treatment.

Patient was given

Tr digitalis M X

Tr Camph Co M 20

Spr Ammon Aromat hourly

Whisky every four hours.

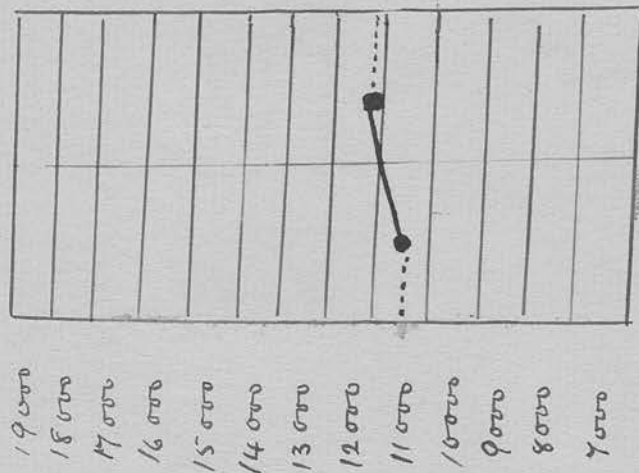
Progress.

The Physical signs remained much the same in the lung, but he gradually got weaker. His pulse became very weak and rapid, and he died on the night of the 15th of March.

A Post Mortem was obtained which confirmed the diagnosis. The whole of the right lung was in a state of consolidation and the lower lobe of the left lung was congested and consolidated inferiorly. The heart showed nothing abnormal except very large antemortem clots.

The Kidneys were slightly congested.

3 4 day of illness.
14 15



LEUCOCYTOSIS CHART

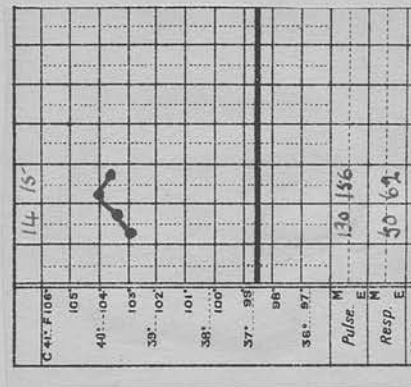
POLYMORPHONUCLEARS	-	78.3.
TRANSITIONALS	-	10
LARGE LYMPHOCYTES	-	2
SMALL LYMPHOCYTES	-	9
MYELOCYTES	.	.5
EOSINOPHILES	.	.2

DIFFERENTIAL COUNT TABLE.

BILOBES
 HORSESHOE
 MULTILOBED

8
71
21.

SHAPE OF NUCLEUS.



TEMPERATURE CHART.

CASE XVII.

Woman Age : 43, unmarried

Occupation : Charwoman

Residence : 5 Vennel

Admitted : February 20th

Examined : do.

Complaint : shortness of breath cough,
spit, and pain in her right
side.

Duration : Seven days.

History

Family history shows a phthisical tendency.

Her surroundings are very poor and exceedingly
dirty. She gets very little food and is
given to taking alcohol in excess.

Present Illness.

Commenced a week ago with a shivering attack.

The pain and cough came on and she got grad -
:ually worse and was admitted on the 20th Feb.

State on Admission.

She is feeble badly nourished woman and when

admitted was in a profound state of neglect. She is slightly cyanosed but shows no herpes nor other morbid appearances.

Temperature 100 .5

Respiratory System.

Shallow respirations 40 per min. very painful especially on coughing.

Spit is rusty and tenacious contains polymorphonuclear cells and Fraenkels pneumococci.

Physical Examination.

Showed a pneumonia of the Right middle and lower lobes, with accompanying pleurisy.

Circulatory System.

The Heart was slightly enlarged on right side. The sounds closed with the first sound somewhat flapping. Pulse was weak and rapid 124.

Nervous System.

She was quite unconscious on admission and is rather wildly delirious at night.

Urinary System.

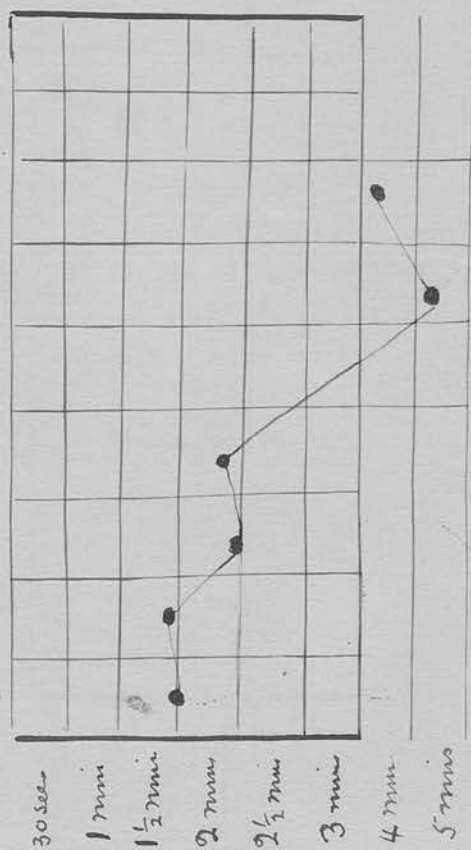
Urine pale straw Reaction acid, specific gravity 1027. Contains no albumen. The

Chlorides are diminished. The other systems showed the usual symptoms of a severe fever.

Progress and Treatment.

She was given Pot Iodid gr V Spr Ammon Aromat M 20 every four hours and Tinct Strophanthus V every four hours.

Her temperature remained about 100 for three days then fell to normal, that is on the tenth day of her disease. Her Pulse fell from 114 to 84. After this she progressed satisfactorily and the lung condition gradually cleared away.



COAGULATION CHART.

	20	22	25-	28	29	30
POLYMORPHONUCLEARS	89	87.4	84	70.5	-	69
TRANSITIONALS	3	2.1	1	2	-	1
LARGE LYMPHOCYTES	6	6	10	11	-	7
SMALL LYMPHOCYTES	2	3.5-	4	15-	-	22
MYELOCYTES	-	1	1	-	-	
EOSINOPHILES.	-	-	-	1.5	-	

DIFFERENTIAL COUNT TABLE

the left side. She took to bed and has been getting steadily worse.

State on Admission.

Well developed woman, with no herpes nor cyanosis. She has signs of advanced venereal disease about the vulva.

Temperature 103.

Respiratory System.

Respirations 50 per min and are hurried and laboured. She has a short cough but no spit.

Physical examination.

Showed consolidation of the left lower lobe with bronchial breathing, medium crepitations, and increased vocal resonance. Coarse friction was heard at the upper border of this and in the axillary region. The lungs were otherwise normal.

Circulatory System.

Heart normal, Pulse 150 per min low tension, and poor volume with no thickening of artery wall.

Her urine was straw in color acid in reaction

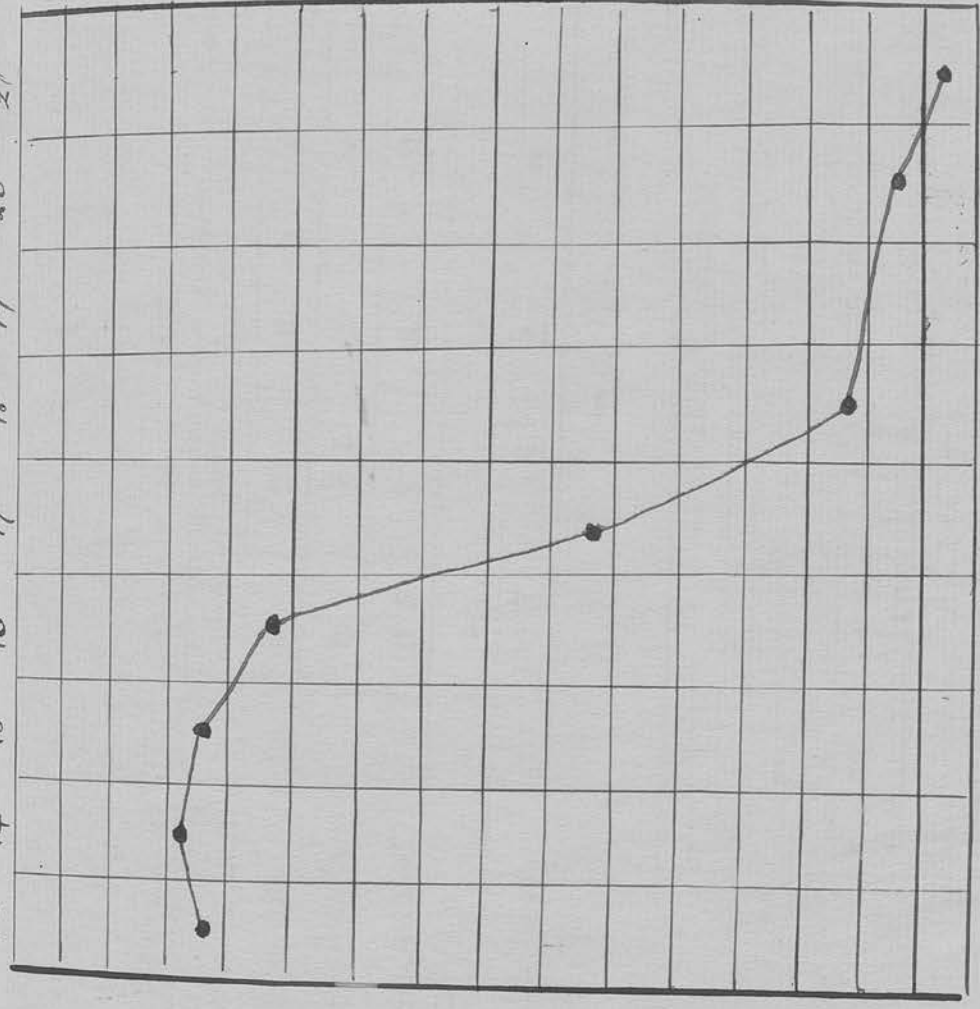
Specific gravity 1020 and showed a deposit of mucus. No albumen was present.

Progress and Treatment.

She was given Pot Iod gr V with Spr Ammon Aromat M 30 every four hours with Tinct Strophanthus M V every four hours and whisky a tablespoonful every four hours. She got rather worse her respirations being 60 on the 15th and the dullness somewhat increased in area. On March 16th she had her crisis and the temperature fell from 102 to normal. Her pulse however and respirations still remained very rapid. The Strophanthus was changed for Tr Digitalis for the next day or two they remained very rapid and then she commenced to recover and progressed satisfactorily.

5- 13 6 7 8 9 10 11 12 13 day of leucocytes

22000
21000
20000
19000
18000
17000
16000
15000
14000
13000
12000
11000
10000
9000
8000
7000

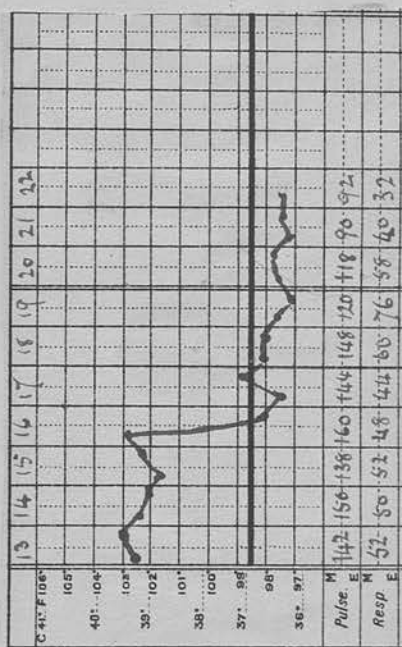


LEUCOCYTOSIS CHART.

TEMPERATURE CHART.

DIFFERENTIAL COUNT TABLE.

	13	14	15	19
Polymorphonuclear cells	80.1	81	80	65
Transitional cells	3	2	2	1
Large Lymphocytes	6	8.2	10	7
Small Lymphocytes	10.2	8	6	25
myelocytes	-	-	-	-
Eosinophile cells.	.7	.8	2	2



CASE XIX.

Man Aged : 21

Occupation : Cabman

Examined : 27th February

Complaint : pain in left side with
cough and sticky spit.

Duration : four days.

History

His home is quite comfortable and he gets plenty of good food. He is very moderate with alcohol. Naturally much exposed with his work.

Present Illness.

Started four days ago. He came in that night feeling chilled and next morning he had fever and a pain in his side. He gradually got worse and was seen by me on the 27th of January.

State on Admission.

Well developed man with good muscularity. He is perspiring freely and shows some herpes on

his lips. A malar flush is present but no cyanosis.

Respiratory System.

Breathing 35 per min, hurried and costo abdominal in type, Cough short, and very painful. Spit orange in color very viscid and contains pneumococci.

Physical Examination.

Showed consolidation of the whole of the right middle and lower lobes with bronchial breathing and medium crepitations. Vocal fremitus was diminished and absent in the lower region. Vocal resonance was increased over the middle lobe but absent from the lower.

Friction was heard in the mid axillary line extending round to the infra spinal region.

Circulatory System.

The Pulse was slightly dichrotic and showed the venous wave otherwise the circulatory system was normal.

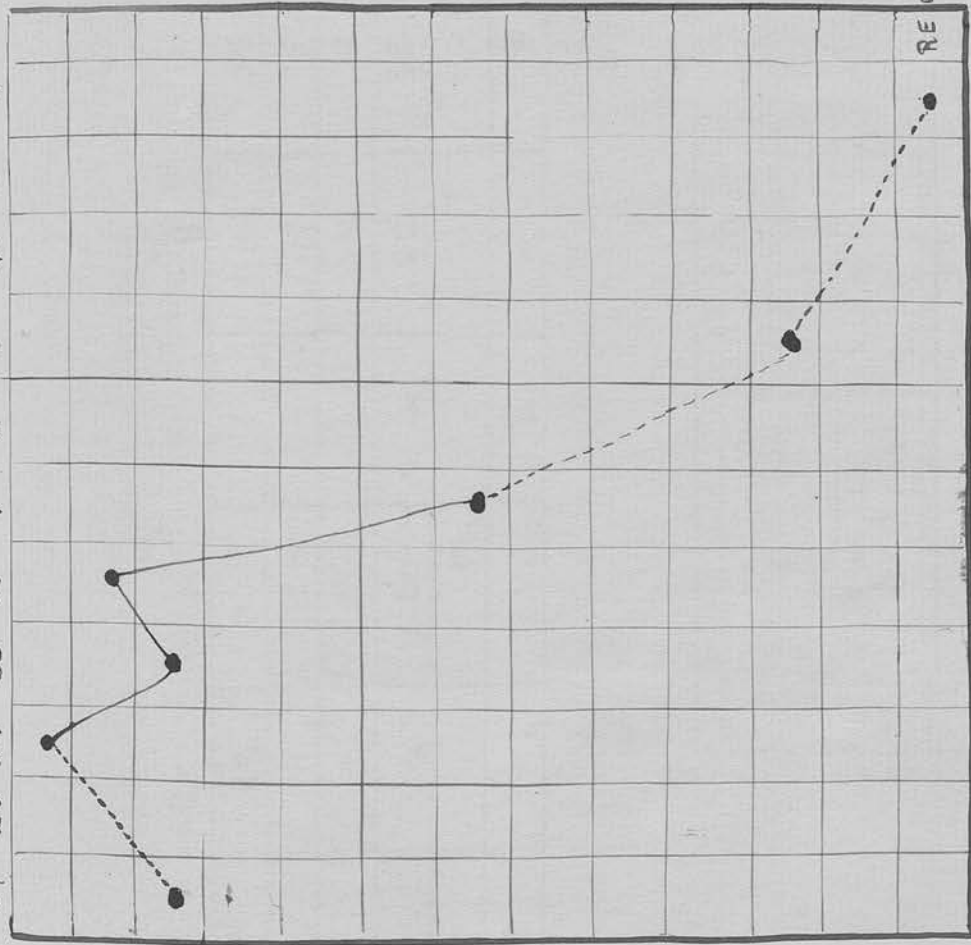
Progress and Treatment.

He was given Pot Iodid gr VII. t i d Spr

Ammon Aromat every four hours. Kept
in bed and on a milk diet. On the 2nd he
had his crisis, that is on the eighth day
of his illness. After this he progressed
to recovery without any complications.

day of illness

20000
19000
18000
17000
16000
15000
14000
13000
12000
11000
10000
9000
8000



CASE XX.

Man Age : 18
 Residence : Canongate
 Occupation : Labourer
 Admitted : 20th February
 Examined : do.
 Complaint : pain in left side and
 cough with spit.

History, family :

No hereditary tendencies.

Personal :

Well fed and is a teetotaler. His home is comfortable five living in one house of two rooms. His work is exposed and hard.

Previous Illness.

He was dismissed from the Army five months ago on account of heart trouble as he calls it. Otherwise healthy.

Present Illness.

Four days ago he was engaged in a wrestling bout and as soon as he got home a severe chill developed. It was succeeded by the stabbing

CASE XX.

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Residence : Canongate

Occupation : Labourer

Admitted : 20th February

Examined : do.

Complaint : pain in left side and
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History, family :

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Personal :

Well fed and is a teetotaler. His home is comfortable five living in one house of two rooms. His work is exposed and hard.

Previous Illness.

He was dismissed from the Army five months ago on account of heart trouble as he calls it. Otherwise healthy.

Present Illness.

Four days ago he was engaged in a wrestling bout and as soon as he got home a severe chill developed. It was succeeded by the stabbing

pain in his left side more marked posteriorly -
he got worse until admission.

State on Admission.

He is a well developed strong lad height 5 ft.
4 in. Weight 10 stone. He is rather flushed
but neither cyanosed nor does he show any herpes.

Temperature 104 .4

Respiratory System.

His breathing was 32 per min regular and shallow
and costo abdominal. There is a short cough
and a sticky white spit muco-purulent in
character and contains Fraenkels pneumococci.

Physical Examination.

Chest is well formed and covered with muscle
and moves equally on respiration.

The lungs appear normal except at the left
base where there is a dull area from the lower
border of the lung to the lower angle of the
scapula. High pitched bronchial breathing
with a few fine crepitations are heard here.
Vocal fremitus and vocal resonance are both
increased. Above this and extending somewhat

anteriorly medium friction is heard, over the rest of the left lung the breathing is rather coarse and an occasional rônchus is heard.

Circulatory System.

The area of dullness of the Heart is slightly increased and a slight blowing systolic is heard in the mitral area. The pulmonary second sound is accentuated. In the other areas the sounds are closed.

Pulse rate 84 regular in time and slightly irregular in force. Tension and volume are fair and the artery wall is not thickened. The other systems show the usual changes in pneumonia. There is no delirium and the urine contains no albumen.

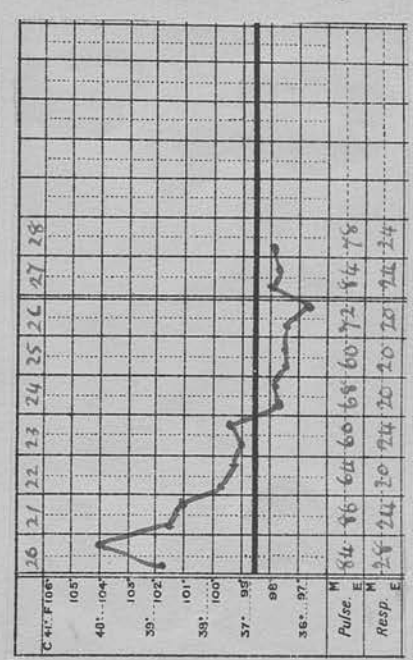
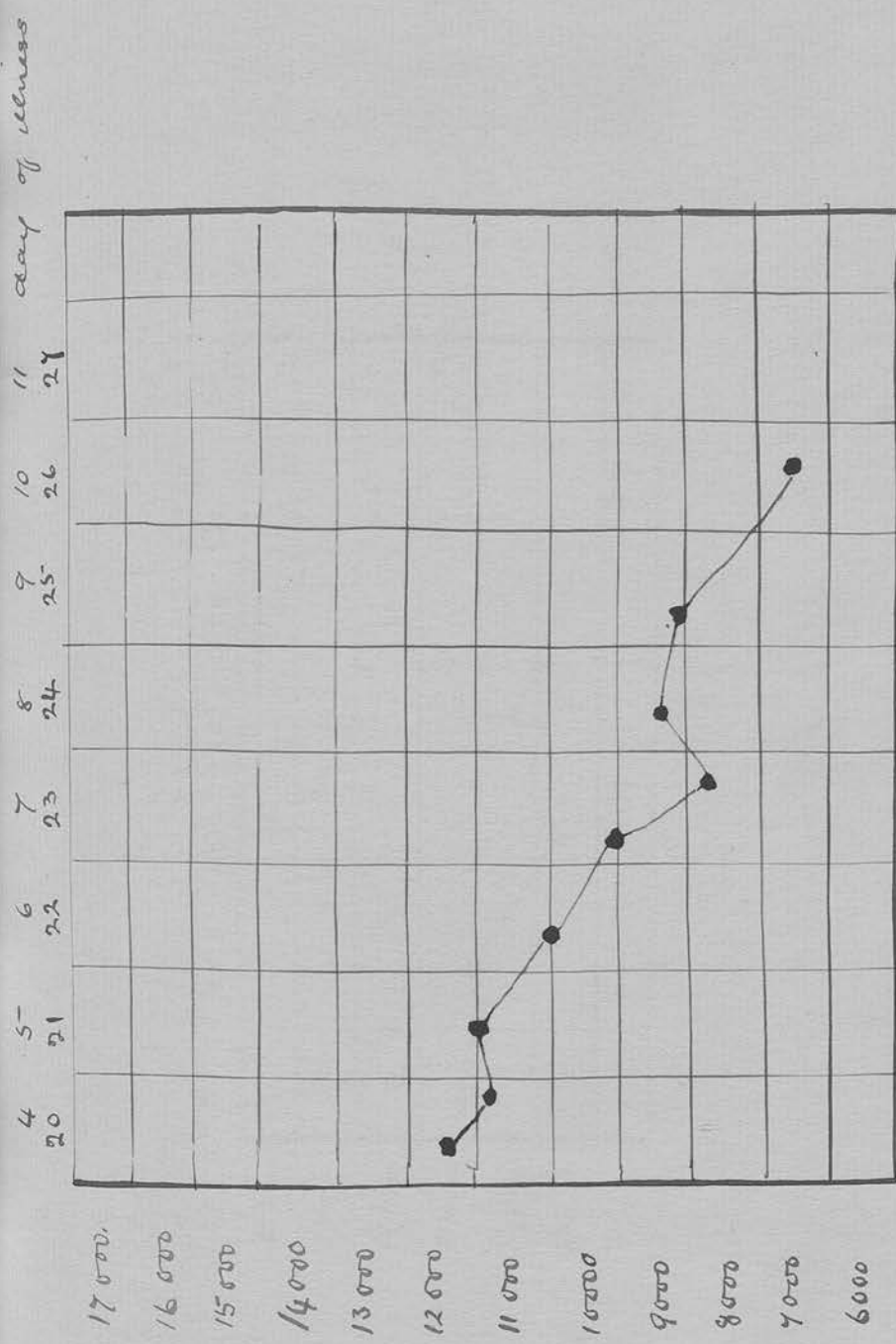
Progress and Treatment.

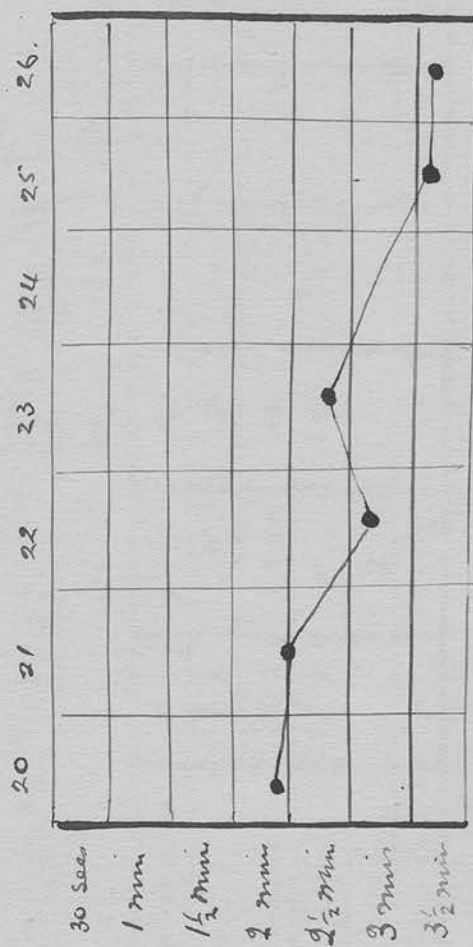
Patient was given Spr Ammon Aromat and Spr Aether Nit every four hours. He was kept on a milk diet. On the 21st, the fifth day of his illness he had his crisis his temperature fell from 104 to 101 and on the following day touched normal. He made a good recovery and the dullness rapidly cleared away the

crepitations became coarse and then subsided
and he was discharged on March 5th.

TEMPERATURE CHART.

LEUCOCYTOSIS CHART





COAGULATION CHART.

	20	21	22	23	25	28	7
POLYMORPHONUCLEAR	71.1	68.3	74	-	-	62.2	-
TRANSITIONAL	7.1	5-	5	-	-	5.1	-
LARGE LYMPHOCYTES	10	12.4	10	-	-	10.2	-
SMALL LYMPHOCYTES	10.1	11	8	-	-	18.5	-
MYELOCYTES	1	2	1	-	-	-	-
EOSINOPHILE CELLS	.7	1.3	2	-	-	4	-

DIFFERENTIAL COUNT TABLE.

CASE XXI.

Pneumonia followed by Jaundice.

Man Aged : 47
Occupation : Ship Engineer
Admitted : 21st February
Examined : do.
Complaint : Pain in side and shortness
 of breath.
Duration : Six days.

History Family:

Is satisfactory. His home is fairly comfortable. His occupation makes him live a very exposed life with great variations of temperature. He has not touched alcohol for many years, and has always been a healthy man.

Present illness.

Early on Saturday morning he woke up with a shivering fit. He got up but had to return to bed. He was feverish had a severe headache and went off his food. He was much troubled with a bad cough, pain in his side and sticky spit. He was better for a day

or two and then got worse and was admitted to the Infirmary.

State on Admission.

Height 5 ft. 8in.

Weight 10 stone

He is not cyanosed, but of a very jaundiced appearance.

Respiratory System.

He had a well formed chest and both sides moved equally on respiration.

Physical Examination.

Showed consolidation of the whole of the right lung with bronchial breathing and some crepitations. On the left side, but for an occasional Ronchus nothing was heard abnormal. Along with the pneumonia he had well marked jaundice. Skin yellow and rather itchy conjunctiva very yellow. High coloured urine showing bile, and pale coloured motions. His liver was slightly enlarged projecting one inch below the costal margin and the dullness superiorly being continuous with that of the consolidated lung.

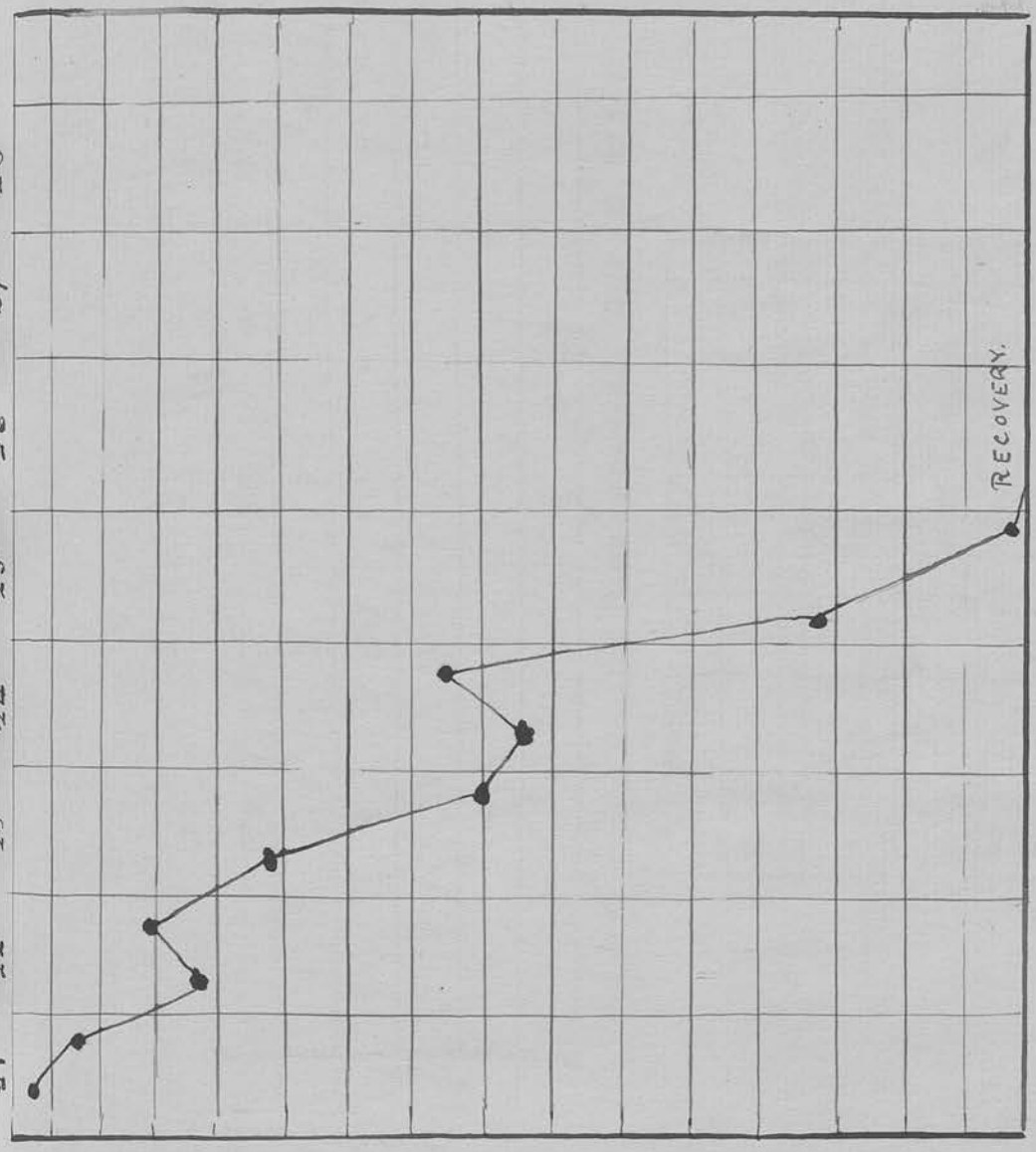
Circulatory and other systems showed nothing abnormal to a typical pneumonia.

The Spleen was not enlarged.

Progress.

On the 21st his temperature came down to normal and remained satisfactory. He made a good recovery the pneumonia clearing rapidly away. The Jaundice remained for a day or two until the 24th and than also subsided.

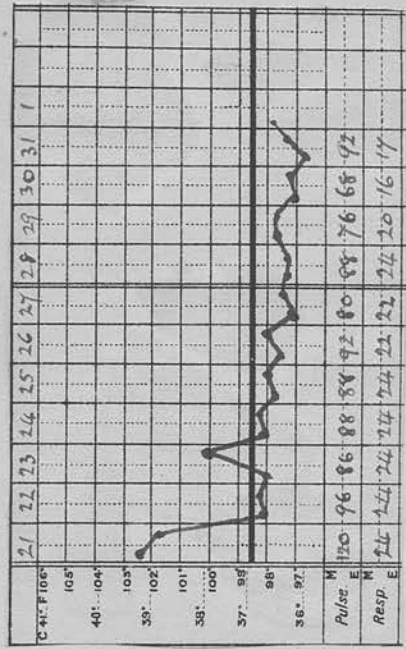
13 day of illness



LEUCOCYTOSIS CHART.

	21	22	23	30
POLYMORPHONUCLEARS	92	87	82	75
TRANSITIONAL	5-	2	1	.4
LARGE LYMPHOCYTES	3	5.5	10	5
SMALL LYMPHOCYTES	4	5-	6	18
MYELOCYTES	-	-	-	-
EOSINOPHIL CELL	.5	.5	1.	1.6

DIFFERENTIAL COUNT TABLE



TEMPERATURE CHART

CHAPTER II.

(1) Leucocytosis of Pneumonia.

It may perhaps not be out of place here to make a very short summary of the work of previous observers on this subject.

Hayem and Gilbert studied two cases of typhoidal pneumonia, and found that the white cells were not increased in number, and that there was no close fibrinous network. They stated that in acute lobar pneumonia they found that the leucocytes

numbered in (1) Slight cases 8000 - 12000

(2) Medium cases 12000 - 20000

(3) Severe cases 20,000 - 36,000

Boekmann observed both the red cells and the white cells, and he found the whites increased in all of his sixteen cases, except two. He found the red cells reduced during the fever and stated that he considered their numbers to be inversely proportional to the height of the fever.

Cabot states that the degree of leucocytosis does not run parallel to the height of the fever, or to the amount of lung involved. He considers

- (1) Mild infection : vigorous reaction - slight
leucocytosis
- (2) Severe infection : vigorous reaction - marked
leucocytosis
- (3) Severe infection : feeble reaction - no
leucocytosis.

Pick noticed that in pneumonia, complicating smallpox, or in those whose resistance is reduced by age, alcoholism or typhoid or chronic disease, there is no leucocytosis.

Limbeck and Pick say that the leucocytosis comes early, and the latter had one case where there was a hypoleucocytosis followed by a marked leucocytosis. They also mentioned that the leucocytes fell before the temperature at the crisis, and that in a pseudo crisis no fall of leucocytosis occurred.

Limbeck stated that in fatal cases a rise in the number of leucocytes occurs before death.

Tumas considers that the leucocytosis outlasts the crisis by one - three days.

Von Iaksch noticed that cases without leucocytosis were almost always fatal and suggested

the use of injecting turpentine to bring on a leucocytosis.

Pichler used pilocarpin or nuclein to induce a leucocytosis, but found that with neither could he be certain of a positive result, and when this obtained, that the degree of leucocytosis was not high. He believes this drug - leucocytosis no gain to prognosis.

Hare and Pichler both found that antipyretics reduce the degree of leucocytosis.

Von Iaksch had sixteen cases which showed leucocytosis - of these three died. Without Leucocytosis he had five cases of which three died, and of these, two were both lungs affected, and the other the entire right lung.

Tschistovitch looks on want of leucocytosis, as a bad sign, and refers deaths with a big leucocytosis to a localization, either in some vital organ or to very extensive infection.

Cabot states that severe cases with a low leucocytosis almost invariably die, and reports an exception of a case by Stockton of a child with three relapses. The first with

4000 white cells

the second with

7840

and the third with

7600

Maragliano also states that patients of his have lived with 4000 leucocytosis and attaches great importance to degenerative changes in the red cells. He demonstrated that the fall in the leucocytosis after antipyretics was due to the dilation of the peripheral vessels.

Lachr finds the red cells and haemoglobin to become reduced during the disease, and rise after the crisis. He had sixteen cases, all had a leucocytosis and two of them died.

Rieder found

(1) That the leucocytosis comes on very early, as he examined three cases, six, fourteen and sixteen hours after the initial rigor, and found a high degree of leucocytosis in all.

(2) That the leucocytosis does not rise towards the end in fatal cases or is only very

trifling.

(3) That there is no parallel between the amount of lung involved and the amount of the leucocytosis.

Monti and Berggrün found that in children, the leucocytosis was highest just before the crisis, and that it increased with the extension of the pneumonia.

Billings had records of twenty two cases, only one of these had no leucocytosis and died. He considered that if both lungs are affected that the leucocytosis is very high.

Bieganski in thirteen cases had eleven with leucocytosis, and two without, and one of his cases had a low leucocytosis to start with, and rose before death.

Ewing in 101 cases found leucocytosis absent in six of them, and these all died.

Stiemon found that the increase in the leucocytosis was due to the increase of the polymorphonuclear cells.

Türk described the increase as due to

I

SEX	AGE	LEUCOCYTOSIS	REMARKS.
M	46	42,000.	
M	24	36,000.	
M	62	32,100	DIED.
M	10	30,100	
M	34	30,000	DIED.
F	43	25,000	
M	30	22,000	
M	46	21,000.	DIED.
M	47	21,000	
M	21	20,000	
M	41	19,800	DIED.
M	48	19,700	
M	19	19,300	
F	24	19,100	on SYPHILIS CASE
M	22	19,000	on phthisis CASE
M	19	12,500	DIED
M	46	12,000	DIED.
M	18	11,400	VERY MILD CASE
M	33	11,300	DIED.
M	64	10,700	DIED.
M	22	7,000.	PAROTITIS DEVELOPED. L. ROSE 521,000.

polymorphonuclear cells, and laid especial stress on the stimulation forms.

Cabot found the polymorphonuclear elements increased. The small lymphocytes diminished. The eosinophiles diminished during the disease, and increased after the crisis, and myelocytes occasionally present. In all the large number of cases he has studied 11.9 was the highest count recorded.

Bieganski found in fatal cases without leucocytosis, that the polymorphonuclear cells were decreased.

Rieder found them increased.

Billings finds them normal.

PERSONAL OBSERVATIONS

In order to condense my results as much as possible, I have made out a number of tables. TABLE I. shows the greatest number of leucocytes recorded in each case, with the age, sex, and result.

Of the cases below 12,500 - four died out of six, while above this number, four died out of fifteen. It shows that below 19,300 every case

either died, developed a septic complication, or was a pneumonia upon some other illness, as syphilis, or tubercle. The one exception to this is, that of Fergusson a boy of 18 who had a very mild uncomplicated attack.

Two cases in this list are of special interest. The last on the list Tait, a lad of 22, who when admitted only showed 7000 white cells. I made eight counts in two days in order to ascertain that my technique was not at fault, and this was the highest number I recorded. Then his leucocytosis commenced to increase and otitis and parotitis developed. The highest leucocytosis recorded was, 21,000 and was undoubtedly due to these other causes as the pneumonia had almost cleared off.

The other case is that of Wilson, a man aged 33. For two days after admission 7000 was the highest number of leucocytes recorded. Then it ascended and reached 11,300, the day before he died. The Post Mortem showed a large abscess in the right lower lobe.

Now Pick and Bieganski both recorded

similar cases but we have no record of the result of their Post Mortems.

In all other matters I think the table speaks for itself.

II

N	A	POSITION	LEUCOLYTOSIS	
M	46	RIGHT LOWER LOBE	42,000	
M	24	RT middle & LOWER LOBES.	36,000	
M	62	LEFT LOWER LOBE	32,000	D
M	10	Right middle and LOWER LOBES.	30,100	
M	34	Right middle & LOWER CONFLUENT BRONCHOPH.	30,000	D
F	43	Right middle & LOWER LOBES.	25,000	
M	30	LEFT UPPER & LOWER LOBES.	22,000	
M	46	DOUBLE.	21,000	D
M	47	WHOLE RT LUNG	21,000	
M	21	RT. middle & LOWER.	20,000	
M	41	ENTIRE RT LUNG	19,800	D
M	48	RT LOWER LOBE	19,700	
M	19	RT Lower & middle	19,300	
F	24	LEFT LOWER LOBE	19,100	
M	22	LEFT LOWER LOBE	19,000	
M	19	DOUBLE	12,500	D
M	46	WHOLE LEFT LUNG	12,000	D
M	18	LEFT LOWER LOBE	11,400	
M	33	RT LOWER LOBE	11,300	D
M	64	DOUBLE.	10,700	D
M	22	RT LOWER & middle LOBES.	7,000	

TABLE II. shows the leucocytosis in relation to the amount of lung involved and its position.

Cases

RIGHT LUNG AFFECTED	12
LEFT LUNG AFFECTED	6
BOTH LUNGS AFFECTED	<u>3</u>
	21 cases.

Deaths

RIGHT LUNG	3
LEFT LUNG	2
BOTH	<u>3</u>
	8 died

Leucocytosis according to the part of lung involved.
I had no apical cases without the base being involved as well.

A.DOUBLE PNEUMONIA. three cases all died.

Average highest leucocyte

count 14,733

B. WHOLE OF LEFT LUNG INVOLVED. two cases - one died - one lived

Average highest leucocyte

count 17,000

C. LEFT LOWER LOBE. four cases - one died -
three lived.

Average highest leucocyte

count 20,325

D. ENTIRE RIGHT LUNG. two cases - one lived -
one died.

Average highest leucocyte

count 20,400

E. RIGHT LOWER LOBE. three cases - one died -
two lived.

Average highest leucocyte

count 24,333

F. RIGHT MIDDLE AND LOWER LOBES. seven cases -
one died - six lived.

Average highest leucocyte

count 24,800.

AGE HIGHEST TEMP. HIGHEST LEUCOCYTOSIS.

41	104.5	19.800.	D
48	104.3	19.700	
46	104	42.000	
22	104	7 000	
19	104	12.500.	D
18	104	11 400.	
10	103.9	30.100	
24	103.8.	19.100	
22	103.7	19 000	
30	103.5-	22.000.	
19	103.5-	19 300	
34	103.	30.000	D
43	102.9	25 000	
46	102.8	12 000.	D.
24	102.7	36.000	
21	102.7	20 000	
33	102.2	11.300	D.
64	102.	10.700	D.
62	101.8	32 000	D.
47	101.5-	21.000	
46	101.1	21.000.	D.

TABLE III. shows a comparison of the highest temperature recorded, and the highest leucocyte count recorded.

Of 11 cases showing temperature above 103 only two died, while of 10 with a temperature below this, six died. In the two cases I had of men over 60, neither had a temperature over 102 although one had a leucocytosis of 32,000 and the other of 10,700.

As regards the leucocytosis otherwise than that shown in the tables, I found that

(1) Numbers ranged from 7000 to 42,000 and that there were more deaths in cases below 12,000 than above it.

(2) That the leucocytosis was as a rule as high on the day of admission, as just before the crisis, although one or two cases showed a slight rise. In this I differ from the observations of Monti and Berggrün but it must be remembered that their observations were on children and that the only child I had among my cases was a boy of 10 and in him a rise of two thousand took place in three days.

(1)
 (3) That a low leucocytosis may mean a
 (2)
 mild case a very severe case with low resist -
 (3)
 :ance a case very liable to septic complications.

(4) That for a day or so before death there is a fall in the number of leucocytes present. In this the evidence of my cases differs from the evidence of LIMBECK.

(5) That in a case of pneumonia following upon any other serious disease the leucocytosis is often not as high as in an uncomplicated case

i.e. Cases

a.	Pneumonia and syphilis	19,600
b.	Pneumonia and phthisis	19,000
c.	Pneumonia and hemiplegia	10,700

(6) That in a case of empyema following on a pneumonia the leucocytes fall with the temperature, but do not reach normal, and their rise is one of the first indications of a commencing empyema.

(7) That there is no fall or only a slight one with a pseudo-crisis.

(8) That in many cases leucocytes commence

to fall before the temperature at the crisis.

(9) That the leucocytosis reaches normal several days after the temperature does.

The differential counts in pneumonia. It is upon this branch of the subject that I have spent the most labour, and it has been perhaps the most unremunerative. I found nothing sensational, no 80 per cent of myelocytes or large lymphocytes in my fatal cases with low leucocytosis. In fact fatal or successful, I found little difference in this department. I examined 300 leucocytes as a rule for each differential count and a very much larger number if anything of importance was to be determined.

Highest per cent of Polymorphs LEUCOCYTOSIS at time. Coagulation at same time.

95 p.c.	42,000	1 min	
92 p.c.	10,000	1 min	Died
92 p.c.	16,000	2 min	
92	30,000	-	
92	32,000	-	Died
91	19,000	-	
91	11,000	-	Died
91	18,000	-	Died
90	19,000	-	
90	14,000	-	Died
89	25,000	2 min	
87	16,000	2 min	
84	18,000	-	
84	34,000	1 1/2 min	
81	30,000	2 min	
81	22,000	1 1/2 min	
80	9,000	1 1/2 min	Died
80	19,000	-	
80	17,000	-	Died
78	12,000	1 min	Died
74	10,000	2 1/2 min	

As regards Polymorphonuclear cells, I found 95 per cent the largest count and 53 per cent the smallest I made. I also found that in the great majority of cases, that the largest percentage of these cells was on the day of admission, and in four of them the largest percentage was on the day of death.

The conclusions I formed from my cases were the following

(1) That the maximum percentage of polymorphonuclear cells has no bearing upon the termination. In my 21 cases I found that the fatal cases were fairly equally divided one dying with 92 per cent, while a case with a maximum of 74 per cent lived. This conclusion I formed only from the 21 cases shown. I believe that in a case without leucocytosis and a low percentage of polymorphs the prognosis is bad, but I cannot think that the same applies to a case with a large leucocytosis.

(2) That up to the crisis, the usual ratio is that

A. 1. Polymorphonuclears

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(2) That up to the crisis, the usual ratio is that

A. 1. Polymorphonuclears

2. Transitionals

3. Large Lymphocytes are tremendously increased.

4. Myelocytes occasionally increased.

B. 1. Small Lymphocytes

2. Eosinophile cells

are relatively and absolutely diminished.

That after the crisis the polymorphonuclear cells get diminished in numbers, while the large Lymphocytes are rather increased for a day or two, and then they too subside. That an occasional macrophagic cell appears in the circulating blood. That transitional cells and myelocytes diminish while small lymphocytes increase to their normal numbers. Eosinophile cells appear again in the circulating blood but are not increased in numbers.

(3) That in my four fatal cases with a maximum leucocytosis below 12,000 the polymorphonuclear cells are increased although perhaps not quite so much as in successful cases.:-

The exact numbers are :-

Four fatal cases with leucocytosis

below 12,000 average 85 per cent.

Seventeen other Pneumonia cases

87 per cent.

In this I entirely agree with ²¹REIDER. It might be argued that all these cases have leucocytosis but of a low degree, while BILLINGS states that his were without leucocytosis and from them, he inferred that the white cells were normal in number. I have however the two cases of 7000 and below it on admission and they showed :-

90 per cent the fatal case

and 84 per cent the case afterwards followed by parotitis.

(5) That the appearance of eosinophile cells early is a point of good prognosis, as also is that of the macrophagic cells after the crisis, showing as they do, that the toxin is neutralized. The case complicated with tapeworm was of interest in that it showed no increase of eosinophile cells during the pneumonia and after the crisis they were only slightly increased.

(6) That in none of my cases before death did I find an entire loss of granulation as

described by DURHAM. I am however somewhat inclined to agree with HANKIN. I think that in a few of my cases the granulations were not so well marked as in normal cases.

Coagulation time in Pneumonia.

The normal coagulation time as determined by Wright's little apparatus is stated to be 3 mins. Now it is almost universally known that in Pneumonia the extravascular clotting of the blood is very much increased in rapidity. In this differing from the acute exanthemata where clotting is slow or fails to occur (Cabot 1901). It was one of the diagnostic symptoms of pneumonia in the days when the physicians lancet was appealed to, to relieve the patient of his toxic blood. But not only is the tendency to clot externally present in this disease, but to clot internally as well. Naturally one would expect clotting in the lungs where the vascular walls are probably altered, so that a tendency to clot might be established. In the pulmonary veins, pulmonary artery, auricles, ventricles, and aorta, however antemortem clots are

often found. I know that it is stated in the 1901 Edition of Osler's Medicine that antemortem clot is a very rare complication of pneumonia. I spent some time looking over our Edinburgh postmortem records for the past six months, and found that in no disease was antemortem clot so common.

I find that I have notes on sixteen post mortems in cases of pneumonia and that in only four of these is the presence of ante mortem clot not specially mentioned. One of these four cases was, that of a broncho pneumonia which had become confluent and so simulated an acute lobar pneumonia.

I have the coagulation records of twelve cases of pneumonia. All of them taken from day to day or every second day from the time of admission until the patient might almost be said to have recovered.

IV.

AGE Highest Temp. Highest leuc'tosis ^{fastest} Coag time Result.

46	102.8.	12,000	30 secs	death
33	102.2.	11,300.	30 secs	death
64	102	10,700.	35 sec	death
34	103	30,000	35 sec	death
22	104	7000	1 min	recovery
46	104	42,000	1 min	do
10	103.9	30,100	1 min	do
24	102.7	36,000	1 min	do
22	103.7	19,000	1 min	do
43	102.9	25,000	1½ mins	do
30	103.5-	22,000	1½ mins	do
18	104	11,400.	2¼ mins	do.

TABLE IV. shows the highest recorded leucocyte count and the shortest recorded coagulation time of each case given along with the result.

It shows the following :-

(1) That every case with a coagulation more rapid than ~~one~~ minute, died. Of the four fatal cases ~~shown~~, antemortem clot was present in two, and absent in one, the case of John McHardie the confluent broncho pneumonia. In the fourth case no post mortem was obtained but every symptom pointed to its presence.

(2) That the increased rapidity of coagulation does not correspond to the number of leucocytes present in the circulating blood.

Tables IV and VI show this.

To cite a few examples -

Man Case I with 42,000 leucocytes took a coagulation time of one minute, but this was also the time taken

by the blood of another case to coagulate with 10,000 leucocytes. Then in another, we find the blood taking one and a half minutes to coagulate with 9000 leucocytes and taking two minutes with 25,000. Then another case took two minutes to coagulate with 30,000 leucocytes, and another took the same time with but 16,000. In this however Table IV. speaks for itself.

(3) That the coagulation time does not depend to any very great extent upon the percentage of polymorphonuclear cells. Table IV. also brings this fact out.

The blood of the two cases with 95 and 92 per cent took one minute to coagulate, and another case with 78 per cent also took this time.

The third from the top of the column took two minutes to coagulate with 92 per cent while we find one with 80 per cent taking one and a half minutes only and another with 81 per cent the same time.

(4) That the height of the temperature has no effect upon the coagulation time when studying cases collectively, as a case took 30 secs. to

Coagulation.	anti mortem clot
30 secs	Present.
30 secs	Present.
35 secs	No Post mortem
35 secs.	absent.

Coagulation	area of lung involved.
30 secs	whole left lung affected
30 secs	right lower lobe
35 secs	Double pneumonia.
35 secs.	Confluent broncho pneumonia.

coagulate with a temperature of 102, while another took two and a quarter minutes with a temperature of 104, For other examples see Table VI.

(5) That the twelve charts which are inserted along with their respective cases show as a rule there is a gradual shortening of the coagulation time up till the crisis, sometimes more abrupt just before it. Then when the crisis has occurred there is a considerable lengthening of the coagulation time and that it then drops somewhat slowly down to the normal of the individual.

(6) That this curve is not affected by variation of temperature daily or day to day.

(7) That in cases with a fall by lysis the coagulation time also comes more slowly to normal.

(8) That in cases followed by empyema that the coagulation time becomes slower, and after the crisis, and that during the course of the empyema a rise in the coagulation time occurs.

(As I have not seen a fatal case of this complication I am only in a position to record those with a favorable issue.)

Now, if this increased coagulability is not due to the number of leucocytes (as is evidently the view held by CABOT) and does not depend upon the percentage of polymorphonuclear cells nor upon the temperature, then upon what does it depend ? The text books state that it is due to the richness of the fibrin forming elements in the blood. This however is a condition of the blood found in several diseased states and not accompanied by the same rapid coagulation.

Blood plates can hardly be regarded as the cause, for almost all authorities are agreed that they are reduced in number before the crisis, to be increased in the circulating blood after. Now during this time when they are reduced the coagulation time is short.

I am inclined to think that there is a deep reason for this coagulation, and one that is often obscured and this introduces me to the next chapter.

Leucolysis.

Leucolysis in Pneumonia.

Now the presence of this is a debated point.

LÖWIT in 1892 pointed out the presence of a hypo-leucocytosis after the injection of certain substances and stated it was due to Leucolysis.

GOLDSCHIEDER and JACOB agreed with the hypo-leucocytosis but explained it by negative chemiotaxis.

EVERARD and MASSART found that peptone, tuberculin, and certain living cultures were not able to destroy leucocytes in vitro. I would here point out the use of the word "certain" and that the "leucocytes" were evidently those from healthy blood.

This inability to destroy leucocytes by living cultures was denied by

TSCHISTOVITCH who believes in leucolysis.

POPOFF also believes in the destruction of white cells in the circulating blood in certain conditions. Then come two sets of observers with indirect evidence.

LOWY and RICHTER found an increased alkalinity in the blood of rabbits during the stage of hypo -

:leucocytosis and they go on to describe this, as due to the destruction of leucocytes.

CARO and STRAUSS found changes in the blood of patients, whom they considered to be suffering from leucolysis, were not always demonstratable, and this they ascribed to the better regulating mechanism of man.

In a paper published in 1898 they claim to have demonstrated in the blood, during a stage of hypoleucocytosis an albumose reaction. This reaction is never found in normal blood, except in conditions associated with a great destruction of leucocytes, as empyema, purulent bronchitis, pneumonia, etc.

Bolkin in 1895 had demonstrated that "in vitro" leucocytes taken from pneumonia and typhoid cases became sooner disintegrated than from normal blood, when kept for several days in an aseptic condition. LÖWIT believes so strongly in leucolysis that he explains chemio-taxis on this basis and says that it is due to the products of the destroyed leucocytes.

HORBACEWSKI agrees with this, and ascribes the chemio-taxis to the discharged nuclein from the destroyed white cells.

Very few writers have drawn much attention to the destroyed cells themselves. CABOT has a small and very important paragraph which I may be allowed to quote:-

"Frequently in leukaemia and occasionally in other conditions one sees leucocytes apparently moribund. That they are not always artifacts is shown by the facts that in normal blood they do not appear when treated by the same technique, that reveals them in the blood and hardened clot of leukaemic cases, as well as by the fact that BOTKIN and others have produced similar appearances by keeping the leucocytes a few days in an aseptic state."

Personal observations. In my chapter to follow on phagocytosis - how I produced broken down cells artificially will be fully taken up.

In no slides that I have seen of any disease have I ever noticed the great number of broken down

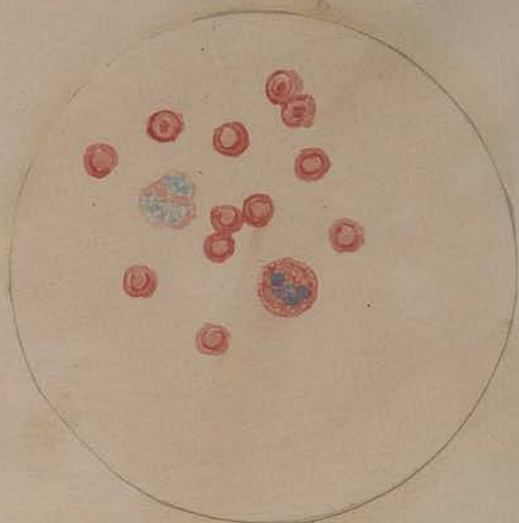


DIAGRAM SHOWING
EARLY STAGE OF
BREAKING DOWN CELLS

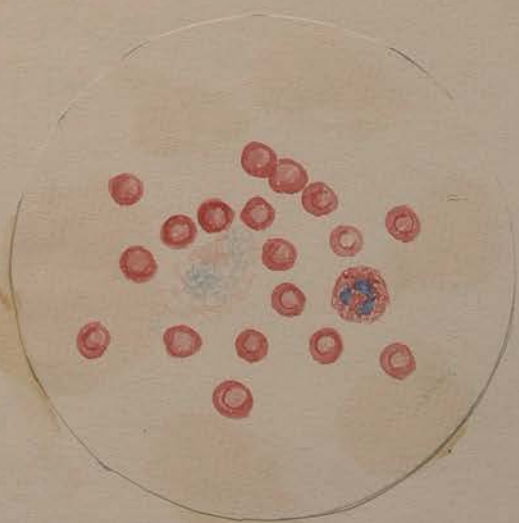


DIAGRAM SHOWING
LATE STAGE OF
BREAKING DOWN CELLS.

cells which appear in some of my slides from pneumonia cases. This could hardly be a technique fault as I usually prepared four films at each drawing of blood and had those of the day before, and day after, to compare with.

I give one or two illustrations of these cells from fields of the microscope. They vary between two extremes a small vacuole in the cell up to nothing remaining but a small mass of pink granules and the faint ragged remains of the nucleus.

In the polymorphonuclear cells the nucleus gets more diffuse, and does not stain so well and may be vacuolated.

The protoplasm becomes vacuolated and finally breaks down into a mass of granules.

The large lymphocytes also undergo a similar change and may look like a homogeneously stained mass of nucleus that has lost its protoplasm and become ragged at the edges (^{ys}KARYOLIS): In the earlier stages it too is vacuolated.

Now these cells are more marked in the severe cases especially in those in which the coagulation time is short.

This has led me to believe that they may be an important factor in the increased coagulation. As these moribund dying leucocytes discharge their fibrin ferment in the circulating blood. And naturally being in considerable numbers, the effect is well marked. I was once asked by one of the leading authorities upon the blood, if, this is so, then why, in a patient suffering from malignant cachexia, where the blood shows many breaking down cells, is the coagulation not only not so rapid but is even slowed ? I answered that injection of nuclein, extract of crayfish muscle, fibrin ferment etc. for some time produced a more rapid coagulation of the blood, but when injected in small doses over a considerable time that the effect was to cause a decrease of the coagulability.

Now I think that Pneumonia and malignant cachexia hold the same relationship to each other. I have mentioned that these broken down cells are more numerous in severe cases of pneumonia. This although generally present is not always so, a few cases where one would expect a large number do not show them. Where the white cells of the

V.

AGE	LEUCOCYTOSIS	SEVERITY	BROKEN DOWN CELLS.
46	40,000	MEDIUM	2 p.c.
18	11,400	SLIGHT	-
33	11,300	SEVERE	6 p.c. DIED.
48	19,100	MEDIUM	3 p.c.
46	21,000	SEVERE	7 p.c. DIED
19	12,000	SEVERE	2 p.c. DIED
24	19000	MEDIUM	4 p.c.
43	25,000	MEDIUM	-
22	Parotidic. 10,000	-	-
33	11800	SEVERE	4 p.c. DIED
64	10,000	SEVERE	2 p.c. DIED.
-	-	-	-

TABLE SHOWING AGE, NUMBER OF LEUCOCYTES, NUMBER OF
BROKEN DOWN AND VACUOLATED CELLS PER CENT OF
POLYMORPHONUCLEARS PRESENT. ON THE DAY
BEFORE THE CRISIS.

blood are normally destroyed, always a much debated point, it is not my object to take up. Suffice it to say that in normal individuals or cases where this organ is working satisfactorily that these cells do not appear in the circulating blood. When it is thrown out of gear either by excessive work or by the action of the toxin, these cells are present.

A glance at the TABLE V~~III~~. will show a few results in this subject. The readings of the broken down cells appear high. The reason of this is, that all polymorphonuclear cells showing vacuolation are included under this head. Of the cases examined on this the day when they are usually most marked four were without them. Three medium cases showed counts of 4.2 and no per cent. The severe cases all died and showed counts on the day before death ranging from 2 to 7 per cent.

Iodophilia.

This subject is the application of the iodine reaction of EHRlich to the polymorphonuclear cells of the blood. It was first brought into prominence by GOLDBERGER and WEISS in the "Wien, Klm. Woch." 1897 No.25. This paper consisted of their observations in cases of local suppuration. KAMMER in the Deut. Med. Woch. 1899 enlarged their observations and mentioned three stages in the appearance of the reaction.

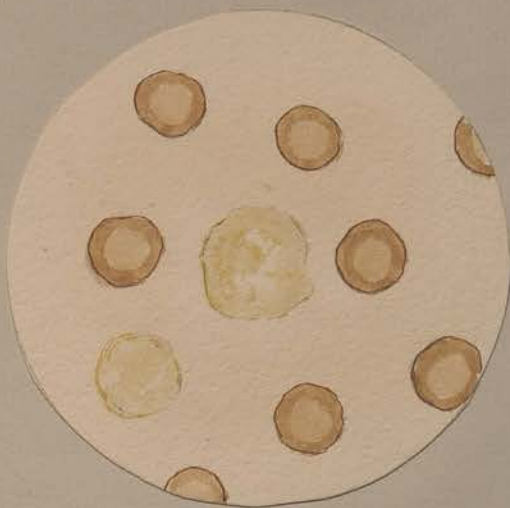
- (1) Slight brownish discoloration of the protoplasm.
- (2) Brown color condensed into very dark flakes and granules.
- and (3) An intense brown discoloration of the whole protoplasm.

I had seen it mentioned in Cabot's "Clinical Examination of the Blood" 1901. that it might occur in Pneumonia so I determined to try it.

My method was as follows :-

I used Goldberger's prescription

Iodi Sublim 1 pt.



NORMAL BLOOD



EARLY STAGE OF
PNEUMONIA.

Pot Iodidi 3 pts

Aq dest 100 pts

Acacia ad Syrupum

Having made my film, I put a small drop on the slide and pressed the cover glass down into it set aside until ready to examine. Among my later cases I modified this somewhat, as I found that the diffuse iodine coloring of the background made this somewhat misleading. I therefore fixed with formalin in alcohol, Gullands' recipe for 5 mins, washed in distilled water then dried, and applied my iodine solution for 10 minutes. I then washed rapidly in water just enough to remove excess dried over a Bunsen and mounted in Canada Balsalm in ~~Zy~~^{yx}sol.

I took the blood films of six healthy students and my own in order to establish a normal I then took films at intervals of a day or two, from twelve cases of Pneumonia, of these, four were fatal cases.

I found that in the normal individual a film is shown in Fig.I that the red cells were stained a pale brown. In some preparations they

were yellowish, but as a rule more of the brown tint.

The polymorphonuclear cells.

These were stained a faint yellow, and contained a nucleus, which was very refractile, and I thought colorless. "Goldberger and Weiss" however thought it citron yellow. Minute refractile granulations could be made out in it. I was unable to find a cell in the normal specimens that contained any brown granules. The small lymphocytes were very difficult to determine. They were also stained a uniform pale yellow much of the same time as the polymorphs, and in the centre refractile looking colorless granules, possibly the nodes of the chromatin network.

Pneumonia Cases -

Red Cells. These as far as could be determined were identical in color with the normal cells. In two of my fatal cases near the close I thought I could make out a somewhat darker tint.

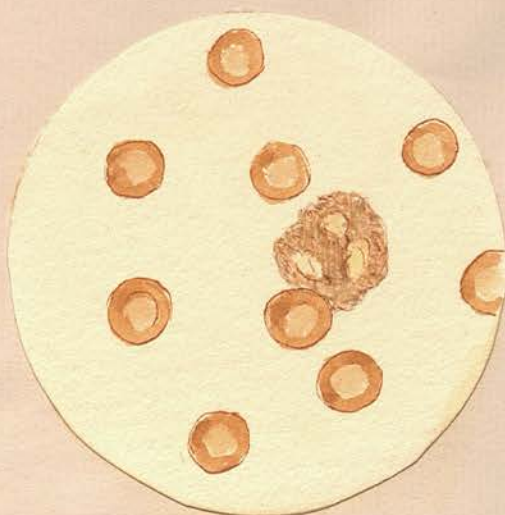
Polymorphonuclear Cells.

These on the first day of admission to

hospital, the third day or so of the disease showed the following changes. Fig. II.

The protoplasm was stained a pale warm brown, rather redder in color than the red cells. Quite distinct from the yellow of the normal leucocytes. It was usually diffuse, and caused the nucleus to stand out either colorless or pale yellow. In two cases very slight granulation of a brown color could be determined. As most often this color was not of the same tint all over the cell, but some portions were considerably darker than others, I was unable to discover any very dark flakes.

The cells showed rather diverse staining power, some being much darker than others. None of them however stained so deeply as the red cells. Just before the crisis in a regular case, the polymorphonuclear cells attained their maximum change. At this time the protoplasm stained brown. Some of a reddish brown, others more of a cold tint. They were darker than they had been before, and in many instances granules of a brownish color could be made out. They were situated all over the protoplasm,



BEFORE CRISIS
IN PNEUMONIA



AFTER CRISIS
IN PNEUMONIA

and in some instances the protoplasm was packed full of them, they did not stand out clearly from the cell, evidently being only a few shades darker than the diffusely stained protoplasm. In size they appeared very small, very much smaller than those of an eosinophile for instance. The nucleus as before was highly refractile colorless or slightly yellow. A great difference was again observed in the staining power of the cells some being many shades lighter than others. I never observed a cell to which I could apply the word intense brown to its color as Kammier did. Those which were most darkly stained being only a shade or two darker in color than the red cells.

After the crisis the brown color remains for a day or two and may even be distinctly present after the number of the leucocytes reaches normal.

Then a larger number of the lighter colored cells appear and the darkly stained ones get fewer in number. The protoplasm of these cells is again irregular in its staining, some parts being darker than others. I also noticed that it is perhaps

darker at the periphera of the cell than elsewhere. This was a fact noticed after an abscess had been opened by Czerny. In my pneumonia cases however it was not well marked and I do not think I would have noticed it but for having my attention drawn to it. Usually in a week or so after the crisis the staining reaction of the cells has again become normal.

The four fatal cases I had, all showed these changes rather markedly. One of them, the case of pneumonia complicated with hemiplegia staining somewhat redder than the others. I took films of all of them within twelve hours of death. In none of these cases however can I say that the protoplasm was stained deeper than at any previous time. In fact rather paler than on the preceding day. In the case of Tait, pneumonia followed by septic parotitis, the reddish brown discoloration of the protoplasm was shown during the pneumonia and instead of clearing away after the crisis, remained, and on the onset of the parotitis was more marked than before, many of the leucocytes showing the brown granules in their protoplasm.

CHAPTER III.

The Phagocytic activity of blood from healthy and pneumonic subjects, when incubated with cultures of Staphylococci and pneumococci.

While reading the British Medical Journal of the 11th January 1902, I noticed a paper by LEISHMAN on the Phagocytic power of the blood. It consisted chiefly of a comparison of the phagocytic power of his own blood and that of a man subject to recurrent attacks of boils. He took a measured amount of blood and added it to an equal amount of normal saline .75 containing living staphylococci Covered with a cover glass and incubated at 98.4 One end of the slide was his own blood. The other that of his patient. When the time was up he slid his cover glass off, fixed and stained by his own method, and then counted the number of organisms in a given number of leucocytes, in both films. He then took the average, and in that way, stated whether the Phagocytic activity of his own blood or that of the patient was greatest. His own blood gave him his normal line. He found that during an attack

of boils the leucocytes of the patient did not take up as many organisms as his own. While after the attack was over, they took up the organisms as well or better than his own.

From the start thus given me, I have worked up this paper. I saw that by diligent work a great deal more could be made out from this than simply the average number of staphylococci taken up by a given number of leucocytes.

The following questions occurred to me :-

1. Can I adopt this to ward work ?
2. Given that the leucocytes take up staphylococci in healthy blood, how will they behave in pneumonia, where the patient is very liable to septic complications ?
3. Given that the leucocytes take up staphylococci in both these conditions, how will they behave to pneumococci, and what difference will there be between normal and pneumonic blood, in their behaviour to pneumococci ?
4. At the different stages of pneumonia how does the reaction of the blood vary ?
5. Is it a true Phagocytosis ?

6. Is there no Leucolysis, and if so does it depend at all on the strength or rather concentration of the microbes or upon the length of time incubated or upon the temperature ?

7. How does the living blood behave with dead cocci ?

8. What changes occur in the cells when taking up the organisms ?

9. How do the different varieties of cells behave ?

10. Are the red cells affected at all ?

My method of Procedure differs considerably from Leishman's, and is as follows :-

I decided that to carry on these experiments I must be as close to my material as possible. I therefore set up my apparatus in the side room of ward 22. The difficulty of these experiments lay in standardising and how I tried to over-come this, will be treated later on. Suffice it to say, that it was only by doing a large number of films at the same time, that I could be certain that they were under the same conditions. The organisms

used were pure cultures of staphylococci, and pure cultures of pneumococci, twenty-four hours old, and grown on blood serum. My diluting fluid was

Normal Saline .75

I commenced as follows :-

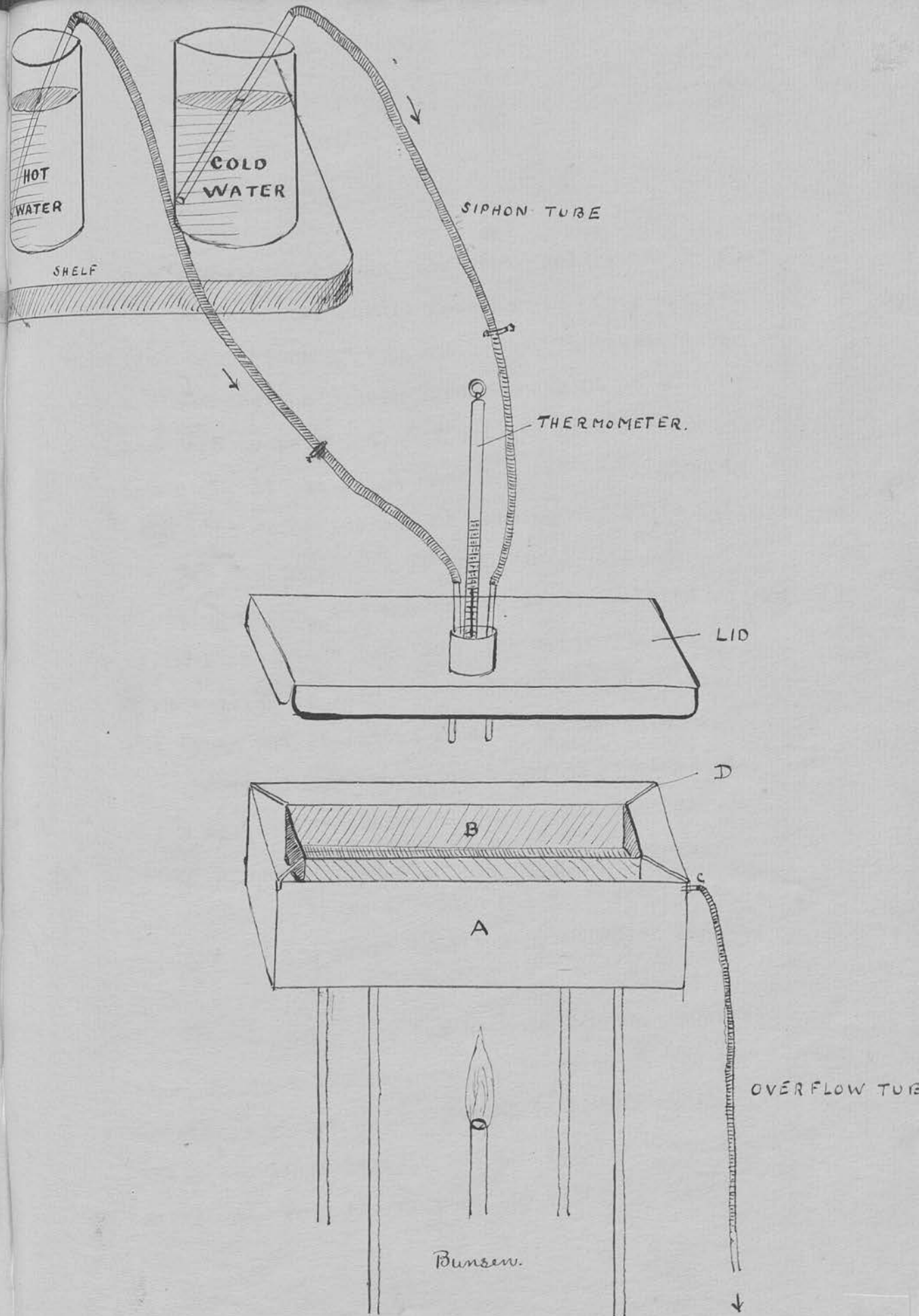
The lobe of the ear, being cleaned with a damp cloth and then dried as recommended by Cabot, without any antiseptics, was punctured with a small lancet and the first few drops wiped away.

The blood is then drawn into a Thoma Zeiss leucocyte counting pipette up to mark 0.5 and immediately emptied into a special small fine glass tumbler that has been specially cleaned. It is capable of containing 2 c.cs. In this is an equal amount of normal saline containing the organisms. This infected normal saline is prepared as follows :-

A given amount is taken of .75 saline solution depending on how many experiments are to be done allowing 0.5 of the Thoma Zeiss leucocyte pipette for each slide to be incubated. Several scrapings of a tube of the pure culture are then taken and

stirred up in the normal saline with a sterilized platinum needle. This must be kept stirred up, and must be made immediately before commencing the experiment. An amount equal to 0.5 of this mixture is taken and added to small tumbler, where it is ready for the blood. When this is added, I make for the side room as quickly as possible and put a drop of this mixture on an ordinary slide, specially cleaned with turpentine and then ether, and then dried. It is then covered with a cover glass, labelled and put into the incubator. Now this incubator for side room work consists as follows :-

An outer tin box A. with an overflow opening at C. Then an inner box B. of a smaller size and not so deep, let in and steadied in position by wires D. It can take out and in, and between these two boxes, is a space which contains the water. Over this fits a lid with a funnel shaped opening in it, which admits a thermometer, a rapidly acting one, and two siphon tubes. These all go down into the water space between the two boxes. The siphon tubes come from glass vessels on the shelf above



one containing hot and the other cold water. The hot water one is hardly necessary. They are for fine variations of temperature. The whole is on a stand above a bunsen burner and kept at a standard temperature. Should it rise a shade too high, it can be at once lowered by running in some cold water instead of waiting for it to fall.

The inner box is then lined with damp blotting-paper, and one or two wooden matches without the heads laid down the centre, so that when a slide is laid in, it does not touch the wet paper but it raised from it by the matches.

The inner compartment should be of a size which will admit twelve slides. The temperature being raised to 98.4 F. it is ready to receive the slides. These are then put in cover glass side up and the exact time taken, at which each is inserted.

When a slide has been in for the required time, it is taken out, carefully dried. The edges moistened with a needle dipped in normal saline. The cover glass is slid off and several cover glass films made from the drop on the slide. Often

they can be slid off without the moistening with saline, but I came to the conclusion that it was usually advisable. The slides are then dried or prepared by the wet method.

The majority of my slides are stained with Eosin and Methylene Blue either being fixed with corrosive sublimate or Formalin and alcohol. Both methods I found brought out the granules of the polymorphonuclear cells well. Some I did with Thyonin blue, others by Grams method and a few by the formula suggested by Leishman.

I may perhaps mention that for every slide incubated with organisms, I made a film with the normal saline, without the organisms, as a test for my technique, and in every batch of slides, I had a few normal blood films under the same conditions as test films.

Now the order I propose to take up is as follows :-

- I. Normal Blood incubated with *Staphylococcus pyogenes aureus* (in these experiments I found thirty minutes the most satisfactory time, so this series I will keep the time limit constant.

Also young cultures of twenty-four hours old were used throughout.)

- a. In moderate concentration
- b. In great concentration
- c. When the Staphylococci have been killed.

II. Normal blood with pneumococci as before time limit constant and young cultures of twenty-four hours used.

III. The Blood of Pneumonic patients with pneumococci. The time limit and the age of culture still constant.

The different reactions obtained at different periods of the disease.

IV. The Blood of Pneumonic patients with staphylococcus pyogenes aureus. Time limit and age of culture still constant.

V. A few remarks on the foregoing paragraphs.

I. Normal Blood incubated with Staphylococcus
Pyogenes Aureus. In moderate concentration.

In my collection of these, I have films from two doctors and several students and have compared the results obtained..

The films were incubated at 98.4 F. and kept in it for 30 minutes.

The results obtained were as follows:-

Polymorphonuclear Cells.

These were in the majority of cases packed with staphylococci. They range from one or two in some cells up to forty and fifty in others. The larger polymorphs contained the more staphylococci and it was in the protoplasm of the cell that they were lodged.

The protoplasm was very vacuolated. The vacuoles being all sizes. Some appeared to be empty, others contain some broken down looking cocci and others the cocci which had just been taken in. Then there were cocci staining very well, lying in the protoplasm of the cell with no vacuolation round them.

The staphylococci were in many instances seen to be double in the protoplasm, and stained clearly with distinct borders.

The neutrophile or faintly oxyphile granules did not take on the stain well. In many cells I was entirely unable to bring them out at all.

The nucleus. In the horse shoe variety. The nucleus in many instances gave me the impression of opening out. The concavity in the middle of the shoe being the part of the protoplasm containing the most staphylococci. The more complicated nuclear forms seemed to take up the organisms better than the smaller varieties as the bilobed. The nucleus stained well, and did not as a rule, show any vacuolation. The polymorphonuclear cells seemed to me to gather near any parts of the film where the staphylococci were more numerous. Though in some places a polymorph could be seen absolutely packed with organisms, and no other cocci to be seen anywhere round it. These were the chief changes I noticed in these cells, there were a few others, but they will be gone into under the staphylococci in great

concentration, as in these films they were comparatively rare.

Large Lymphocytes.

These were distinctly phagocytic. Only the medium sized large Lymphocyte was seen in these normal films, of course, the macrophagic cells proper, being absent. One was seen containing eighteen staphylococci, but as a rule, they contained only four or five cocci. They did not show the vacuolation of the polymorph cells to such a marked extent. In the nuclei the chromatin network seemed somewhat wider than usual.

Small Lymphocytes

These when small sized contained no cocci, that I could be certain of, a few were sticking to their edge evidently by surfaceattraction. At first, with Eosin and methylene blue, I could not be certain, as the cocci were rather difficult to differentiate from nodes, in the chromatin networks of the lymphocytes, which were very darkly stained with the methylene blue. I therefore stained some films by Gram's method using safranin as a contrast stain, and by this method, I could not find any in the

lymphocytes.

In those a little larger, with the zone of protoplasm increased somewhat, the cocci were found, but only in very small amount, only one or so in an occasional cell.

Eosinophile Cells.

These cells seemed to behave in a most uncertain way. A large number of exploded examples were seen, only a mass of oxyphile granules and the fragments of a nucleus remained. I am uncertain whether more were exploded than one would expect, when one remembers that the drop of blood has been gently stirred up with the normal saline and twice been slid off the slide. Others again, had all their granules in situ. I was somewhat uncertain as to whether they showed phagocytic power or not, the very great majority certainly contained no staphylococci. In a few cases I was doubtful, one organism, perhaps two, being just in the situation where there was great difficulty in determining whether they were in the cells, or only lying on them.

B. Staphylococci in great concentration.

In these experiments I again used the same

age of culture and incubated for the same time.

Before taking up the changes in the white cells I may mention the red cell changes. In Section A. the red cells appeared normal but in this Section there was a distinct tendency to laking. The red cells also were slightly swollen. The test films without the organisms appeared normal showing that the fault did not lie in the technique. Now I proved this action of the staphylococcal toxin, in the following way. I added several scrapings of a tube of staphylococci to a little normal saline, boiled it for one minute. Then into four test tubes I put a little of this stock solution, so that tube

(1) was my most concentrated form.

(2) half the concentration of tube 1.

(3) a quarter of the concentration of tube 1.

(4) normal saline boiled for one minute alone.

I then added normal human blood .5 of the leucocyte counter again being used to each of the tubes, and shook them well up. I then allowed them

to stand, having previously taken out of each a known small quantity, which I centrifuged. Both the blood which had stood, and that which had been centrifuged showed the same changes.

The serum was distinctly pink in No.1 tube. It was slightly tinged in No.II. and colorless in III. and IV.

Now for the white cells.

The polymorphonuclear cells showed two classes.

- (1) Those containing staphylococci to excess.
- (2) Those not containing staphylococci or only in very small numbers.

(1) A large number of the white cells 60 per cent. had taken up the organisms. They had engorged them, till some cells looked as if there was not room to get another in. The blue nucleus could be indistinctly seen and a slight cell border all the rest was a mass of darkly stained cocci, which obscured all granulation, and almost hid the nucleus. Some of them were swollen twice their normal size with the cocci.

Then many more had burst. They now looked like a small bunch of cocci, with the remains of a

nucleus somewhere in the middle of them. No signs of the protoplasm could be made out.

Then there was class (2). In this class a number of the cells had taken in one or two staphylococci and others none at all. They both showed almost identical changes. Their nuclei swelled up, became vacuolated and finally broke up, into small filaments, (bearing a great resemblance to pictures by one of our Edinburgh observers of cells he found in fatal cases of Pneumonia without leucocytosis, and which he designates myelocytic polynuclear cells.) Their protoplasm was swollen, vacuolate and contained, perhaps one or two cocci, but in many instances none at all. The protoplasm finally became ragged, and indistinct, and the nucleus broke down, so that all that remained was a cluster of small granules, most of them staining with basophile stain. The large lymphocytes also were in many instances vacuolated and swollen. They contained a few cocci. The small Lymphocytes appeared to be unchanged, and I was unable to find any eosinophile cells intact.

C.Staphylococci which have been killed.

I took several scrapings from a tube of a twenty-four hour old staphylococcal growth, and mixed with normal saline in a test tube, and then boiled for one minute. I then centrifuged, and added a little of the deposit of dead organisms to the fresh normal saline, and blood in my tumbler, and then incubated as before.

The results were as follows :-

- (1) The red cells appeared to be unaffected.
- (2) The polymorphonuclear cells had taken up the organisms well.

The nuclei stained well, and also the protoplasm granulations still were not distinct. Many cells contained forty or fifty cocci, and as before were swollen out with them. In cells which did not contain so many, there was not the same amount of vacuolation, and the cells did not seem to burst so readily. None of the small clumps of cocci round the remains of a nucleus being seen. As before the large lymphocytes contained a few cocci. Several Eosinophiles were seen containing their granules. The young forms of the lymphocyte were

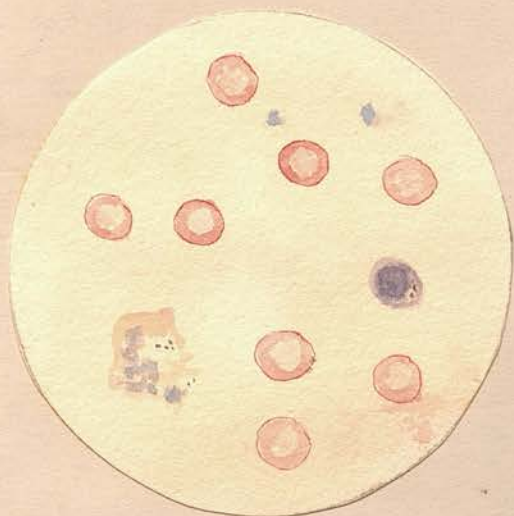
as before not phagocytic.

Now the reason why I did this experiment was the following :-

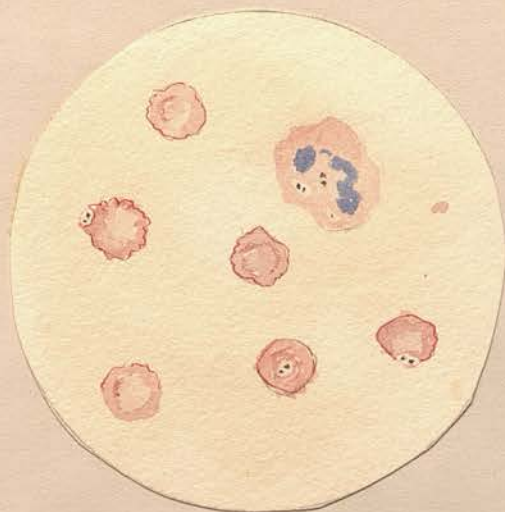
In my experiment A. I found the cocci enclosed, staining well, and in many instances double, as if they were dividing within the cell. I increased the concentration in B. and found that the cocci had so much got the upper hand, that in many instances all that remained of a leucocyte was a small clump of apparently healthy cocci. This rather shook my faith in Phagocytosis, so I carried out these experiments with the dead cocci, and found that the leucocytes engulfed them, proving I think that the attack was on the leucocytes part, not on that of the cocci.



FILM WITH STAPHYLOCOCCUS
PYOGENES AUREUS.



FILM WITH PNEUMOCOCCI



FILM WITH PNEUMOCOCCI
SHOWING CHANGES IN
RED CELLS.

Normal Blood with Fraenkels Pneumococci.

In my experiments with this organism I found that I got somewhat different results with the different ages of culture employed.

The leucocytes taking up a culture of twenty-four hours, somewhat better, than one of two days old. The latter seemed to have more power of poisoning the leucocytes, without being taken up. With very old cultures, five and six days, I found them fairly well taken up by the polymorphonuclear cells, and not so many broken down cells appearing.

In my first series, with a twenty-four hour old culture incubated for thirty minutes at 98.4 I got the following results :-

Red Cells. The chief action appeared to be a tendency to form crenation of the reds. The pneumococci were very often seen to be lying on the indentation formed by the crenation of the red cell. Then the pneumococci, also, were continually seen in the concavity on the surface of the red cells, with an appearance of swelling of the capsule. There was not so much laking as with the staphylococcal cultures.

Polymorphonuclear Cells. These took up a few organisms, not many like the staphylococci, but usually only one or two diplococci, perhaps one might hold six or seven, but this was the great exception. The cells as a rule contained one diplococcus, and were in a very active state, the nucleus was somewhat spread out to the periphery of the cell and the diplococcus usually enclosed in a large vacuole, perhaps two might be in one vacuole, many were seen to be broken down, and half absorbed, while others were quite healthy looking. Then there were cells which were evidently poisoned, and then nuclei became diffuse and not staining well. They then became vacuolated, and broke down, the protoplasm also doing so, and as with the staphylococci nothing but a small mass of granules remaining. Some of these breaking down cells contained a diplococcus, others no trace could be seen of them. In this, it was seen that in moderate concentration the pneumococcus resembled the staphylococcus in great concentration. The former being by far the more powerful in its leucolytic action. As before, I found the granules of the polymorphonuclear cells

not staining nearly as well with eosin, as the films without the cocci. This is simply a confirmation of what Durham alleged occurred before death in some infective conditions, and what Hankin has noticed to occur in leucocytes, when in the presence of microbes. Hankin seemed to regard them as of bactericidal substance. Anyhow I certainly noted that in cells enclosing a diplococcus, and this situated in a vacuole, showing that the cell was in an active state of secretion, that the granules were very indistinct.

The large lymphocytes were in many instances vacuolated, and contained one or two diplococci. Three was the largest number that I counted, in one of these cells. They also in a few instances were seen to be breaking down, but not in the same numbers as the polymorphonuclear cells.

The small lymphocytes were to all appearance unchanged, and no enclosed organisms could be made out in the smaller variety. In some slightly larger, with a ring of protoplasm round, I have undoubtedly found some enclosed diplococci.

The Eosinophile cells as before appeared

uncertain in their behaviour. I have seen them after this incubation with pneumococci with all their granules intact, but only in a very few instances. The very great majority 80 per cent. I should think, having broken down, and their position only marked, by a few granules, and a detached part of nucleus here and there.

III. The Blood of Pneumonic Patients incubated with Pneumococci and compared with normal Blood films treated in the same way.

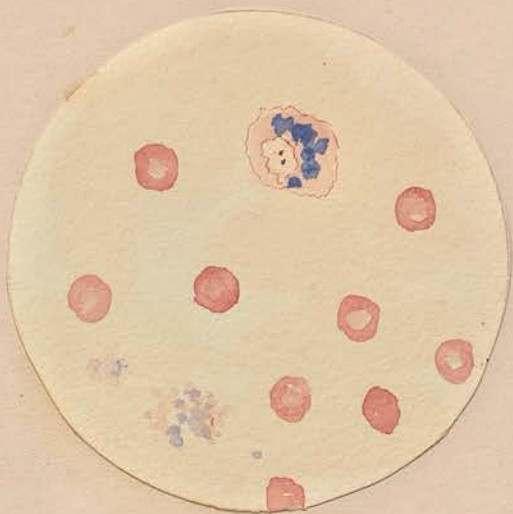
In this branch of the subject I used
three fatal cases
one complicated with empyema
and eight normal cases.

I shall first take up the normal cases.

Before the crisis - These were all cases showing a leucocytosis of from 12,000 to 30,000.

I found that as compared with the normal blood that the white cells did not take the cocci up so readily as the normals. Pneumonia however is a disease which is stated not to attack Doctors, students and nurses so readily as other people, so we may assume that their blood has a certain immunity. I therefore also took the blood of one or two patients not suffering from pneumonia and incubated it along with the pneumonic blood.

The changes in the polymorphs were very marked, vacuolation and breaking down. A much



CELLS BROKEN DOWN
AFTER INCUBATION WITH
PNEUMOCOCCI



Do.

larger number were found to be in a broken down condition, than those in the normal blood.

12 per cent in this as compared with 8 in the normals, with the same concentration. It was especially in the young varieties that this was noticed. The transition cells, and those whose nuclei were of the bilobed type, suffering most - while many polymorphs with multilobed nuclei, stained well and appeared quite normal. A smaller percentage of polymorphs took up the diplococci than in the normal blood, as a rule. They contained from one to four diplococci. I also thought that the red cells were smaller and more crenated than in the normal films, but a comparison of this sort is very difficult to decide upon. Maragliano however, stated, he found degenerative changes in the red cells of severe pneumonia cases. The films taken after the crisis, I expected the white cells would show a greatly increased phagocytic power. I found however that this was not so. On comparison with normal films, they were very much alike and if anything, I should say that the cells in the normal blood took up a larger number of

diplococci. The broken down cells were far more plentiful, shortly after the crisis, than later on. The large lymphocytes contained only in a few instances diplococci.

I must here mention the behaviour of the very large lymphocytes called macrophages I only found one or two examples in my films, though I picked cases to show them. These were extremely phagocytic containing many pneumococci, in one, I counted nineteen diplococci,

The behaviour of the eosinophile cells, I was not able to determine, as none of the cases showed them in sufficient quantities, to make it, at all certain.

I must make a remark about the fatal cases, they were three in number. Two had leucocytosis,

30,000 and

13,000

The other had only a leucocytosis of 6000.

The first two agreed in all particulars with the normal cases, but the leucocytes of the 6000 case, did not take up the diplococci as well, and showed far more broken down ^{cells} ~~films~~ than the other two.

IV. The Blood of Pneumonic Patients when incubated with *Staphylococci pyogenes aureus*.

This series of slides were somewhat interesting. Before the crisis the polymorphonuclear cells behaved much as in normal blood. The transition forms and those with a more simple form of nuclei were not nearly so active, as the multilobed variety. The more mature forms however took up the staphylococci well, fifty-two being counted in one of these. There was comparatively little leuco-lysis even among the immature forms. I made one or two films however, very concentrated, corresponding to section I.B. In them few leucocytes except small lymphocytes remained. The great majority being in a state of granular disintegration, or replaced by small clumps of cocci. After the crisis the cocci were not taken up well at all, an average of six per polymorphonuclear instead of eight before. I took the blood of a boy with acute septic parotitis following pneumonia, and incubated it at the same time. His phagocytic power was low, but was not lower than two ordinary pneumonia cases of the same date, without complications. The other four had a higher phagocytic power.

The points I have remarked most from the experiments are the following :-

(I) That the taking up of the cocci is evidently an act of Phagocytosis on the part of the leucocyte and that they exhibit this to a much greater degree with Staphylococci than with pneumococci (and also slightly more in normal blood than in that of pneumonic patients.)

(II.) That these cocci when engulfed in the leucocyte call forth an active secretion, causing small vacuoles in the cell, and that they are gradually disintegrated and dissolved in these vacuoles.

(III) That under certain conditions, the cocci have the power when engulfed of poisoning the leucocytes and causing them to disintegrate. This is more marked with pneumococci than with staphylococci and is accentuated by the following conditions.

(A.) In pneumonia when young stimulation forms of leucocytes are present.

(B.) When the resistance of the leucocytes is lowered by. (1) increased temperature that is above normal 98.4 but practically this only becomes

apparent when the temperature is above 106. F.

(2) Keeping the leucocytes for a longer time in the presence of the cocci.

(C) When the cocci are in great concentration or in certain virulent cultures as some show this change much more than others.

(D) Technique faults as

(1) Too long an interval between the withdrawal of the blood and admission to the incubator.

(2) Sliding the films off the slide when too dry. The reason for this is that vacuolation seems to precede disintegration and naturally a vacuolated leucocyte is more liable to damage than a normal one.

IV. That certain cultures have the power of causing a few leucocytes to disintegrate although no cocci are contained in the cells.

C O N C L U S I O N .

I must now draw this Thesis to an end. I have tried to state my facts as clearly as possible, and in one or two places have attempted to show the direction, the evidence, of these cases would point to.

I shall be happy, if I have added my mite, towards the literature of the immense subjects, which have been dealt with.

The blood is a very large and comparatively new subject. It is one, that instead of dogmatising upon insufficient data, as I am afraid has already been too often done, the observer should content himself by adding his observations to the already existing knowledge, and leave his mind open, to the new facts, which are continually being brought out, as methods of procedure are improved upon, or new ones invented.

I shall therefore leave to others, the work of drawing their own conclusions from the facts herein set forth. I have only once attempted a

possible theory founded upon my own observations, and upon those of others. That theory is, that the moribund, breaking down, leucocytes, in the blood in cases of Pneumonia by discharging their fibrin ferment, while circulating in the blood, may be an assisting factor, in the greatly increased coagulation found in Pneumonia.

With these very few remarks I therefore finish this Thesis.
